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The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society.

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President of the American Academy of Periodontology

1951-1952

Dr. James E. Aiguier of Philadelphia, Pennsylvania, was installed as President of the American Academy of Periodontology at its annual meeting in Washington, D.C., on October 11-13, 1951. This honor culminates a long period of service for Dr. Aiguier to the Academy of Periodontology and to dentistry.

Since his graduation from the University of Pennsylvania in 1917, Dr. Aiguier has been active in many dental organizations as a member, committeeman, or officer. It is impossible to list all of his activities and honors, but noteworthy are: Chairman of the Publicity Committee, 7th International Dental Congress; Chairman of the Section on Periodontology, American Dental Association, 1949; Member of the Committee on Dental Preparations, National Formulary, 6th and 7th Edition; and Member of the Executive Committee of the Pennsylvania Emergency Child Health Committee, 1933-1942. He is a member of Sigma Xi, Omicron Kappa Upsilon, and Delta Sigma Delta.

Members of the American Academy of Periodontology anticipate a successful year under the able leadership of Dr. Aiguier.

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THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-TWO

Occupational Bruxism and Its Relation to Periodontal Disease

by JOHN I. INGLE, D.D.S., M.S.D., *Seattle, Washington*

BRUXISM has long been considered in a general sense as the habitual grinding and gritting of the teeth. It is seen quite frequently in restless children and is thought then to be associated with such diverse conditions as tooth eruption, vesical irritability and intestinal parasites. The adult who is afflicted is usually considered to be a "nervous" individual. Frequently this grinding habit is carried on throughout the day (bruxomania) and night (bruxism), though it is often a feature of the sleeping hours alone. It may be associated with various anxieties, with overwork, complete concentration or unrealized aggression. Two variations in the actual condition are observed. The patient may fully triturate all the teeth with a complete masticatory stroke or may grind only on one point of premature tooth contact, unconsciously attempting to wear this prematurity down and thus achieve full occlusal contact. A habit of either type inflicts very obvious damage upon the morsal surfaces of the teeth as well as upon the periodontium and the temporal-mandibular joint. This then constitutes the common "neurosis" type of bruxism.

Bruxism of occupational derivation is seen less frequently but must, however, be considered as a possible damaging factor in periodontal diseases. Miller[1] and Salzmann[2] have thoroughly discussed this condition and have stressed its etiologic importance to the periodontist. We all have observed the "bobby pin centrals" of the hair stylists, the notched anteriors of the roof shingler, and the damaged periodontium of the thread biter. While these damaging habits are not strictly bruxism in its pure form, they achieve a destruction comparable to bruxism. The case of occupational bruxism to be discussed in this paper lies beyond the realm of habit or nervous grinding.

CASE REPORT

Mr. J. S. G. presented on December 10, 1949 for routine periodontal treatment. The history revealed that he was a fifty-one year old farmer of English parentage. His medical history was negative, his diet excellent and his appearance that of a healthy, robust individual who had spent a great deal of time out-of-doors. The chief complaint was, "loose upper front teeth." These teeth had begun to noticeably loosen about ten years previously and the condition

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Fig. 1. The severe morsal wear and steep incisal guidance exhibited in the case of Mr. J. S. G. is well illustrated in the initial photograph.

had gradually worsened. The patient's general dental treatment had been extensive, but in a periodontal sense had been limited to an annual prophylaxis.

EXAMINATION

Visual examination revealed changes in both the hard structures and the supporting tissues. The most striking feature was the extreme wear to the morsal surfaces of all the teeth (Figs. 1, 2). Upon close inspection of the soft tissues, inflammatory redness, flabbiness and swelling of the free gingiva surrounding the upper anterior and bicuspid teeth was apparent. Hemorrhage, suppuration and cyanosis was also present in these tissues (Fig. 3). Generally speaking, the remainder of the mouth appeared to be in good periodontal health.

Examination with a periodontal probe revealed a pocket depth in the affected regions of 4 to 7 mm. and, in addition, a 4 mm. pocket between the lower central incisors. In some areas moreover, the depth would have been greater if gingival recession had not occurred beforehand. There was a bifurcation involvement of both upper first bicuspids. Subgingival calculus was present but not extensive, and the *involved teeth all exhibited third degree mobility.*

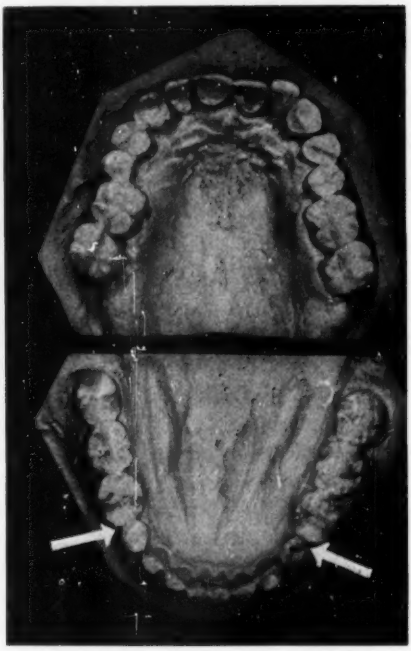

Fig. 2. Study casts demonstrating the overall morsal wear causing the flattened cusp height in the posterior and steep incisal guidance in the anterior. The mandibular second bicuspids (arrow) are congenitally missing which accounts for the occlusion of the lower third molars against the upper second molars.

Radiographs (Fig. 4) were taken and study models (Fig. 2) made at the first appointment.

DIAGNOSIS

From the accumulated evidence, a diagnosis of traumatism was made. The conclusion was reached that such extreme wear of the teeth as existed was undoubtedly the primary factor leading to the destruction of the periodontium. The posterior teeth were worn flat to the point where a cusp height was practically non-existent. By contrast, in the anterior mouth a deep overbite had caused the teeth to wear on a *steep* plane rather than on the *flat* plane exhibited in the posterior mouth.

It would now be beneficial to digress momentarily to consider how destruction of the anterior periodontium may be related to such extreme morsal wear. Hanau, in his laws of articulation,[3] has clarified the relation of cusp height to incisal guidance. He has stated that a flattened cusp height in the posterior should quite naturally be accompanied by a flattened incisal guidance in the anterior. In this particular patient we have noted that the reverse situation exists; namely, a flattened posterior cusp height but a steep incisal guidance. Referring again to Hanau's law, it becomes apparent that the incisal guidance in the anterior must in some manner be flattened to accompany diminishing cusp height. In the untreated case this anterior flattening is accomplished by the natural forces of occlusion which literally thrust the upper anteriors out of the dental arch. Obviously extreme periodontal destruction must follow in the wake of such forceful labial movement. Where our patient was concerned, however, the mere observation of extreme wear and periodontal damage did not of itself provide the answer to the problem of what caused the abrasion.

Contrary to expectations a continued questioning of the patient yielded no positive history of nervous bruxism. This fact was puzzling. Furthermore, no compensating eruption had taken place so that a free-way space of 8.5 mm. was present

Fig. 3. Clinical photograph taken at the first appointment. Note the extreme overbite, the elongated upper left lateral incisor, localized recession and severe inflammation present.

rather than the customary 3.5 mm. interocclusal space. Because of the wide free-way space, it was felt that the patient was constantly clenching the jaws thus preventing a compensating eruption. Yet whenever the patient was observed unaware, the mandible was being carried at rest position. Though the cause of the morsal wear remained a mystery, the attention was turned to a plan of treatment and prognosis.

PLAN OF TREATMENT

The history and examination led to one conclusion only where treatment was concerned; if the upper anterior teeth could not be retained, then all of the maxillary teeth would have to be sacrificed and a full

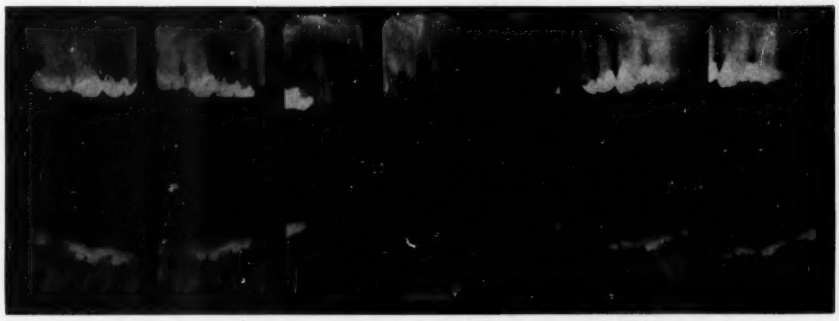

Fig. 4. Initial radiographic study. The level of alveolar bone in the mandible and posterior maxilla is comparatively normal, whereas the alveolar destruction in the anterior maxilla is quite apparent. Observe the bifurcation involvement of both upper first bicuspids.

Fig. 5. Photograph of the case immediately following gingivoplasty in the lower anterior and odontexesis of the remainder of the teeth. The temporary acrylic splints (arrow) have been cemented in place to restore the occlusal vertical dimension and relieve the traumatism in the upper anterior.

Fig. 6. The permanent removable partial denture splint. The appliance restores the occlusal vertical dimension, replaces the missing bicuspids, flattens the anterior incisal plane and splints all of the maxillay teeth as a single unit.

upper denture constructed. The patient's determination to save his teeth if at all possible was a very influential factor in favor of their retention.

The principal problem to be solved was the establishment of a new occlusal level which would alleviate the severe labial thrust on the upper anterior eteth. If this could be accomplished, the long range prognosis of the case would be favorable. The decision was made to construct a prosthetic device to establish this new occlusal level. However, before such an appliance was begun, preliminary surgical procedures were indicated. These would involve the removal of the two upper first bicuspids with bifurcation involvement (whose periodontium had been severely damaged by traumatism complicated by a pipe smoking habit) and the curettage of the upper anterior areas. Additional preparations involved:

a. Full mouth odontexesis
b. Minor gingivoplasty between the lower central incisors
c. Wax records of the occlusion
d. Casts mounted on an adjustable articulator.

TREATMENT

With the articulator as a guide, a new occlusal level was established by opening the incisal pin a full 4 mm. The upper posterior teeth were waxed-up to this new level upon the articulator. Subsequently acrylic splints were cemented to place on these posterior teeth (Fig. 5).

Mr. J. S. G. was thoroughly instructed in the home care procedures and was then dismissed for a two-month trial period. The primary aim was to have the patient experience this new occlusal vertical dimension before any extensive rehabilitation was undertaken.

He returned on February 14, 1950 very well pleased with the new dimension and reported relief from trauma on the upper anterior teeth. The decision was made to proceed with treatment. The carious areas were restored and the occlusal surfaces of the lower teeth were reshaped with improved anatomy and a narrowing of the buccal-lingual dimension. The periodontal surgery was completed* and the two bicuspids were extracted. The areas of surgery were allowed to heal for ten days before proceeding with further treatment.

The temporary splints were removed and new impressions were taken. The case was remounted on the articulator and a new occlusal level was established at the point

*Surgical technique to be covered in a later issue of the Journal.

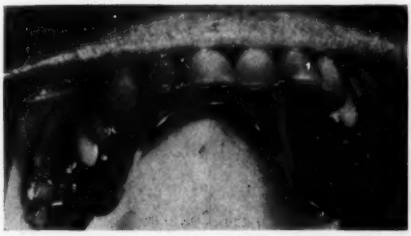

Fig. 7. The removable appliance in place. The posterior teeth are actively clasped and the anterior teeth are passively retained. The thickness of metal in the posterior and the new flattened incisal "table" are apparent.

Fig. 8. Clinical photograph taken at the time of the first recall (three months). Recession, induced by surgical curettement in the upper anterior, has reduced the sulcus depth to an average of 1.5 mm. The labial splinting pins pass through slight notches cut into the incisal edges of the upper central and lateral incisors. The cuspids are splinted from the distal labial (arrow).

where the incisal edges of the anterior teeth just met in the protrusive movement. This step brought about a reduction of the free-way space by 4.2 mm. A permanent prosthetic device had by this time been proven a feasible objective. By its nature, this appliance would be required to accomplish the following:

a. Restoration of the missing bicuspids
b. Restoration of the occlusal level and occlusal vertical dimension
c. Establishment of a flat incisal plane
d. Splinting of all teeth in the arch

The finished appliance covered the occlusal surfaces of the upper posterior teeth and actively clasped all of them. The upper anterior teeth were passively retained from the labial as well as the lingual. They were covered by a lingual "table" which served primarily as a means of establishing a new and flat incisal guidance. Following minor adjustments, the patient was then dismissed on a three month recall.

Upon his return, we were pleased to note that the patient's reaction to this extensive procedure was most gratifying. The gingival tissue was now healthy and firm. The original pockets were reduced to the depth of the normal sulcus. *The upper anterior teeth had tightened from third to first degree mobility.* The new occlusal level was especially appreciated by the patient who had begun to experience temporal-mandibular joint disturbance before the correction (Figs. 7, 8, and 9).

CAUSE OF THE MORSAL WEAR

It was during the recall examination that the cause of the occlusal wear was discovered. The patient was being demonstrated to a postgraduate group studying problems in occlusal equilibration. One of the dentists participating in the course exclaimed that he had seen dozens of cases of this type of wear and that it was caused by riding a tractor.* This proved to be exactly correct for the patient had for a number of years farmed in Central Washington and had spent a great many hours and days driving a caterpillar-type tractor. Through further inquiry it was learned that this type of occlusal wear is very common in these large farming communities and that the dentists of the district see such cases quite frequently.

The wheat farms in this area are measured by the thousands of acres. Heavy-duty diesel tractors are the common power unit used to pull the massive farm equipment. It is quite routine for a farmer to spend twelve hours a day driving the jolting, pounding tractor. Evidently it is commonplace for such a man to unconsciously clench the teeth together to prevent the constant, jolting strain to the

*The author is indebted to Dr. W. J. F. Tretner, Colfax, Washington for this observation.

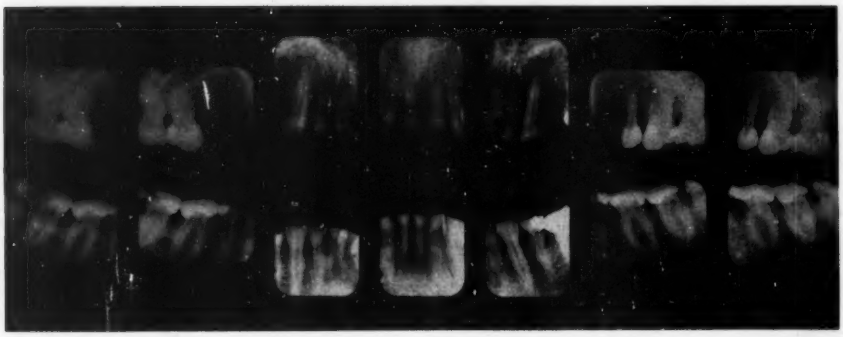

Fig. 9. Recall radiographs taken at 18 months. The upper anterior teeth have tightened from third degree to only first degree mobility. Thickened periodontal membrane of the lower central incisors indicates excessive pressure on these two teeth. This was eliminated by judicious grinding.

mandibular musculature. The entire farming operation is surrounded by a cloud of dry, powdery dust which acts as an excellent abrasive agent in the destruction of the morsal surfaces. If the man is fortunate to have little or no overjet and overbite in the anterior, all the teeth will be worn down evenly with the occlusal pressure being exerted along the vertical axis of the teeth. But if the patient has a decided anterior overbite, the wear will result in damage such as described in this case. Where extreme anterior overjet exists, an impingement of the upper lingual or lower labial tissue by the anterior teeth of the opposite arch is unavoidable.

DISCUSSION

It would seem reasonable to assume that the type of occupational bruxism herein described might also be experienced by military tank drivers and tractor drivers on highway construction crews. Possibly a rubber bite plate such as an orthodontic positioner could be used as a preventive measure. Where extensive damage has occurred the prognosis of the case would of necessity depend upon the severity of the problem presented. Occupational bruxism may take many forms. It would be well for dentists to recognize the importance of that bruxism which is seen in the mouths of men who are operating heavy, mobile machinery in a dust-laden atmosphere.

SUMMARY

1. A case has been described involving severe abrasion of the morsal surfaces of the teeth with accompanying periodontal damage.

2. Occupational bruxism was shown to be the underlying basis for the above described condition.

3. Severe occlusal wear among drivers of heavy, mobile machinery may be considered as one form of occupational bruxism.

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Age Changes and Sex Differences in the Clinically "Normal" Gingiva

by FRANK M. WENTZ, D.D.S., M.S., ALEXIS W. MAIER, M.D., D.D.S., *and* BALINT ORBAN, M.D., D.D.S., *Chicago, Illinois*

INTRODUCTION

THE histologic examination of gingival biopsies has been increasingly employed in the detailed diagnosis of gingival disease. Intimate knowledge of the histologic structure of clinically "normal" gingiva of various age groups is an indispensable prerequisite for a correct histopathologic diagnosis. Such details, however, have not been precisely described or correlated.

It is the purpose of this study to investigate age changes and sex differences in the human gingiva and thus attempt to establish the basis for a better understanding of this structure.

REVIEW OF LITERATURE

The literature contains only a few references concerning the surface appearance and histologic structure of the normal gingiva. King[1] referred to the orange peel appearance of the normal gingiva due to numerous regularly distributed small depressions. Orban[2] stated that usually only the attached gingiva is stippled and he interpreted stippling as an expression of functional adaptation to mechanical impacts, due to the collagen fibers of the lamina propria which elevate the surface. Histologically, he correlated the free gingival groove with the division of the free and attached gingiva. Zach[3] conducted histologic studies of 20 biopsy specimens of clinically normal gingiva. He noted marked differences in both the epithelial and connective tissue elements. In the epithelium he found variations in thickness; relative size of component layers; degree, type or absence of keratinization; parakeratosis; and uniformity of surface. In the connective tissue he emphasized the distribution of the papillary portion according to size, the presence of clearly defined lamina propria, the type of fibrillar structure, the cellularity, vascularity, and the extent of round cell infiltration. Most of his specimens showed a deviation in one or more histologic aspects from the classical description of normal gingiva.

Aprile[4] studied 8 biopsies of clinically normal gingiva from individuals 12-54 years of age and noted histologic variations in relation to age and sex. She stated that the gingiva in the male and female are similar in individuals under 18, but that later female epithelium tends to be thicker. The connective tissue showed no sex variation, however. Infiltration of inflammatory cells was apparent in the connective tissue of all cases. Bernier[5] also noted an infiltration of inflammatory cells in every specimen of his collection of more than 7000 biopsies.

Glickman[6] observed a trend toward diminished keratinization occurring in normal gingiva with increasing age.

In contrast to the scant literature on the age changes in the gingiva, there are numerous reports on detailed studies of the aging of human skin. Primarily, age changes were noted in the elastic fibers. Since the gingiva does not possess elastic fibers, only a relative comparison between these two tissues is possible. MacLeod[7] found the epidermis to be thinner in old age, but failed to give evidence. Ejiri[8] counted the number of layers of epidermal cells in young and old cadavers. He found that with age they

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diminish in number on the head and face, but increase on the dorsal surfaces of arm, hand, and foot. He also noted atrophy of the rete ridges in old skin. Weidman[9] stated that the epidermis undoubtely undergoes atrophy with advancing age. However, he does not clearly define what he considers atrophy.

Ceresca,[10] investigating skin sections and counting the number of dermal papillae per linear unit, reported a moderate diminution in old age. Becker,[11] in a study limited to infants, found a tendency of the skin to be thinner at 6 months than at birth. Atsuki[12] concluded that changes in skin thickness depend upon many variables, age seemingly being the outstanding one; in general, adults have a thicker skin than infants. Evans, Cowdry, and Nielson[13] measured epidermal thickness from basal to granular layers. They indicate a difference of 6.5 microns in comparing epidermis of old and young individuals. Old skins also have a tendency to lose a layer of granular cells and their stratum corneum appeared to be thinner. Hill and Montgomery[14] stated that the condition of the stratum corneum is not influenced by age. The epithelial ridges show atrophy with advancing age. Changes in the arrangement of collagen fibers show no definite relationship to age or region.

MATERIAL AND METHODS

This study is based on the examination of gingival biopsy specimens of 38 female and 64 male patients ranging from 4 to 80 years of age. Approximately an equal number of patients for each decade of life was obtained in order to investigate possible trends in histologic characteristics of the gingiva in relation to age and sex.

These examinations were conducted at the Chicago State Hospital.*

Most of the subjects were permanent inmates of the institution. Mouth hygiene was fairly good. The facilities of an excellently staffed and completely equipped modern dental clinic are available to these people.

Clinical Examination. The clinical examination of the oral mucous membrane and the gingiva was mainly confined to the anterior region of the oral cavity.

Only those patients were selected whose gingiva presented clinically a very low index of gingival inflammation, and who thus fulfilled the following requirements of the so-called normal gingiva; Maier and Orban.[15]

Color	pink
Marginal contour	normal or slightly receded
Papillary contour	pointed
Texture	stippled in most instances
Consistency	firm
Exudate	none
Pockets	none or shallow

The medical history of each patient was studied in detail. Generally, their present state of physical health was fairly good. Previous diseases, as well as function of heart, lungs, kidneys, liver, etc., were recorded. Twenty-one patients were alcoholics with a history of intemperate use of alcohol. Their mouth hygiene was very poor and sometimes showed gross neglect. Examination usually revealed heavy deposits of subgingival calculus. Their gingival tissue, however, seemingly did not keep pace with the existing pathologic conditions. The free gingiva showed very little inflammatory reaction and the attached gingiva presented a definite pallor and firm consistency. In practically every case of

*We should like to express our sincere appreciation to Dr. Edward F. Dombrowski, Superintendent of the Chicago State Hospital, for his permission to use the facilities of this institution. Our thanks are also extended to Dr. Isadore Spinka, Clinical Director of the Chicago State Hospital, as well as to Dr. Victor L. Griseto, Dental Consultant for the Northern District, Department of Public Welfare, State of Illinois, for his whole-hearted cooperation and assistance. Mrs. Rosella Noonan, registered nurse, rendered us great service in preparing patients for clinical examination.

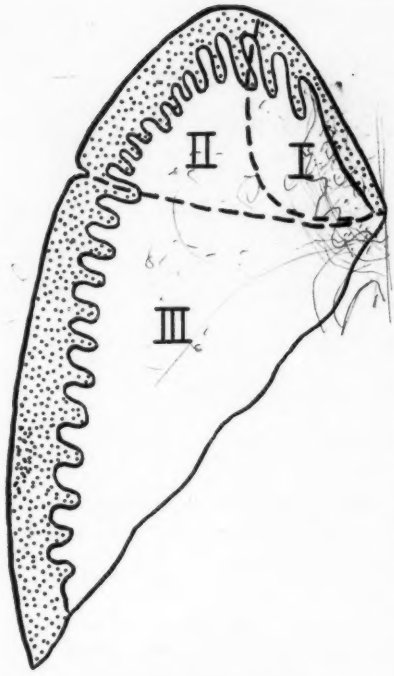


Fig. 1. Diagram of the gingival biopsy showing division into three zones. Zone I—area of gingival sulcus. Zone II—area of free gingiva. Zone III—area of attached gingiva.

advanced alcoholism it was possible to repeat this observation.

A clinically normal gingival papilla in the upper or lower anterior region of the mouth was selected for the biopsy. Color photographs preceded the operation.

Each biopsy was fixed in freshly mixed Zenker-Formol solution (concentration 9:1), embedded and sectioned in paraffin and stained with hematoxylin eosin.

HISTOLOGIC FINDINGS

The gingival biopsy was divided into three zones (Fig. 1):

a) Zone I—The area of the gingival which extends from the depth of the sulcus to the tip of the papilla.

b) Zone II—The area of free gingiva which extends from the tip of the papilla to the region of the free gingival groove.

c) Zone III—The area of the attached gingiva extending beyond the free gingival groove.

Infiltration of inflammatory cells in Zone I, the region of the gingival sulcus, was present in all specimens and a constant finding. Zone II showed this infiltration occasionally. The inflammatory cells of both zones consisted generally of a few polymorphonuclears near the epithelium with plasma cells and lymphocytes deeper in the connective tissue. Zone III, the region of the attached gingiva, was selected for this study because it was not affected by possible modifying factors of microscopic inflammation. All findings reported hereafter will be confined to area III, the attached gingiva.

THE EPITHELIUM

Hornification. Ninety-four of 102 specimens demonstrated complete hornification, predominating over islands of parakeratosis. Often keratinized and parakeratotic areas alternate for some distance.

Eight specimens showed parakeratosis only, without alternating areas of complete hornification. Five of this group were younger than 18 years.

Complete lack of hornification was not observed. In general, complete hornification is a structural characteristic of the gingiva of all ages.

A granular layer was usually not present in the parakeratotic specimens. Seemingly, a granular layer is not indispensable in the formation of the hornified layer since approximately one-half of the specimens with hornification had a granular layer and one-half did not. The hornified layer tended to be thicker in the specimens with a granular layer than in those without a granular layer.

Sex and age did not influence the type of

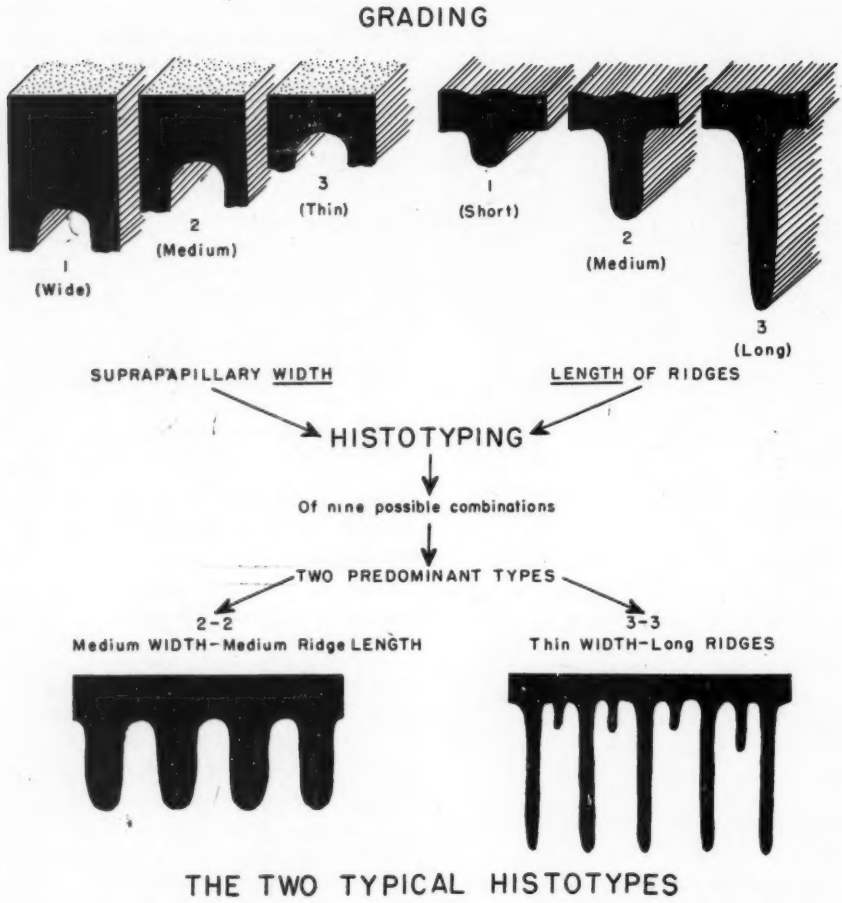


Fig. 2. Diagram showing method of grading and histotyping the epithelium of attached gingiva according to architectural structure.

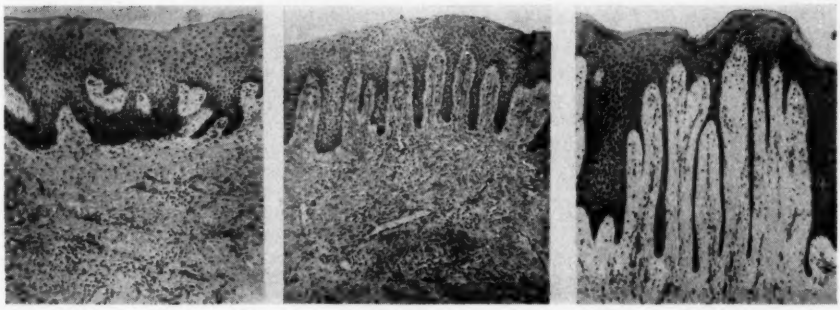
hornification or thickness.

Suprapapillary Epithelial Width. The distance between the surface of the epithelium and the epithelial connective tissue junction at the tip of the connective tissue papilla was considered as suprapapillary epithelial width. It was graded as wide (average 225 μ); medium (average 150 μ); or thin (average 75 μ) (Fig. 2).

Thick. Thick epithelium was noted only in one case, an individual aged 11 years.

Medium. The majority of gingival biopsies showed a medium width. In the 4-14 age group, a medium width of epithelium was found in 10 of 11 cases. In the higher age groups two-thirds of all cases were of a medium thickness.

Thin. A thin epithelial type appeared for the first time in the 15-29 year group, and constituted approximately one-third of the specimens of this group and higher age groups.



A. B. C.

Fig. 3. Photomicrographs of biopsies of attached gingiva showing epithelial histotypes. (A); histotype 1:2 (wide suprapapillary epithelium and medium ridges (B); histotype 2:2 (medium suprapapillary width and medium ridges) (C); histotype 3:3 (narrow suprapapillary width and long ridges).

Length of Epithelial Ridges. Epithelial ridge length was graded as short (average 225 μ); medium (average 450 μ); long (average 750 μ) (Fig. 2).

Short. Short ridges were found in 6 of 11 cases in the 4-14 age group, but were not noted in any of the later ages.

Medium. Medium epithelial ridges were found in all age groups. From age 15 to 80 more than half of the cases were of this type.

Long. A long type of epithelial ridge in a significant number appeared for the first time in the 15-29 year group. It was a constant type and totals somewhat less than one-half of all the types.

Epithelial Histotypes. The factors of epithelial width and length of epithelial ridges enter into typical combinations.

If an epithelial width designated as wide, medium, or thin is given a respective number 1, 2, and 3, and the same numbering is applied to short, medium, or long epithelial ridges a 2-2 combination would indicate a medium epithelial width and medium ridge length, a 3-3 thin suprapapillary epithelium and long ridges. The specimens, when classified in this manner, showed that a 2-2 type and a 3-3 type constituted the greatest proportion. Thus, a definite order of combination is apparent leading to the designation of a definite configuration of histotype of epithelium (Fig. 2). A modification of either one of the numbers was evident; however, it was not significant as a general histotype except a 1-2 combination (short ridge, medium width found only in young age groups).

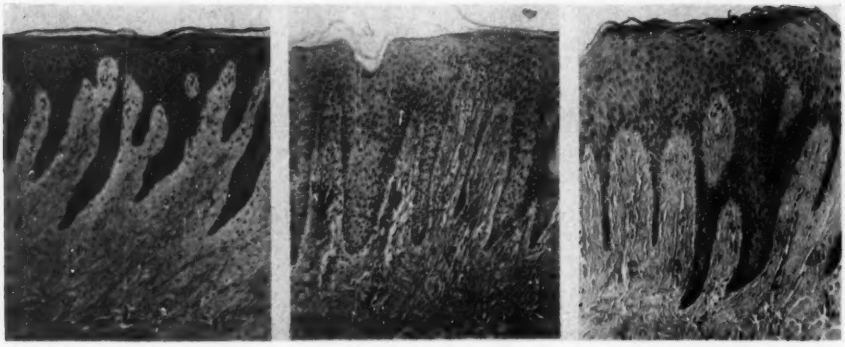
4-14 Age Group. Four histotypes were apparent as 1-1, 1-2, 2-2, and 3-2.

Five of 11 biopsy specimens of this group were of the 1-2 type (Fig. 3a). Four of 10 specimens were of a 2-2 type (Fig. 3b). Generally, a short ridged and medium width epithelium type and a medium ridged and medium width epithelial histotype were predominant in this group.

15-29 Age Group. Three histotypes were prominent in this age sector, a 2-2, 3-2, and 3-3. A trend toward sex differentiation was also suggested. In the male group, 10 of 19 specimens fell into the 2-2 designation and 7 of the 19 were of the 3-3 type (Fig. 3c). Two of the cases fell into an intermediate 3-2 category.

The 3-3 thin epithelium and long ridge histotype found in the male group was without parallel in the female group.

Considered in total numbers, male and



A. B. C.

Fig. 4. Photomicrographs of biopsies of attached gingiva showing connective tissue histotypes. (A); histotype 1:2 (fine textured papillary layer and irregular textured reticular layer) (B); histotype 2:2 (irregular textured papillary and reticular layer) (C); histotype 3:3 (coarsely textured papillary and reticular layer).

female, the 2-2 histotype was found in the greatest number (23 of 33). It is characteristic for females of this group.

30-44 Age Group. The 2-2 and 3-3 histotypes were maintained as well as established types. A sex differential, apparent in the previous age group was lost. More than one-half (18-30) were 2-2 in character, 10 of 30 were of the 3-3 histotype.

45-80 Age Group. More than half (15-27) were of the 2-2 character. Nine of the 27 represented the 3-3 type. The remaining ones were scattered in 2-3, and 3-2 designations.

CONNECTIVE TISSUE

The connective tissue of the attached gingiva is classified as a dense matte type. However, variations in the arrangement of the collagenous fibers were noted:

a) *Finely textured:* consisting of unbundled, diffusely arranged, fine fibers.

b) *Irregularly textured:* consisting of unbundled fine fibers and coarsely bundled fibers.

c) *Coarsely textured:* consisting of bundled coarse fibers.

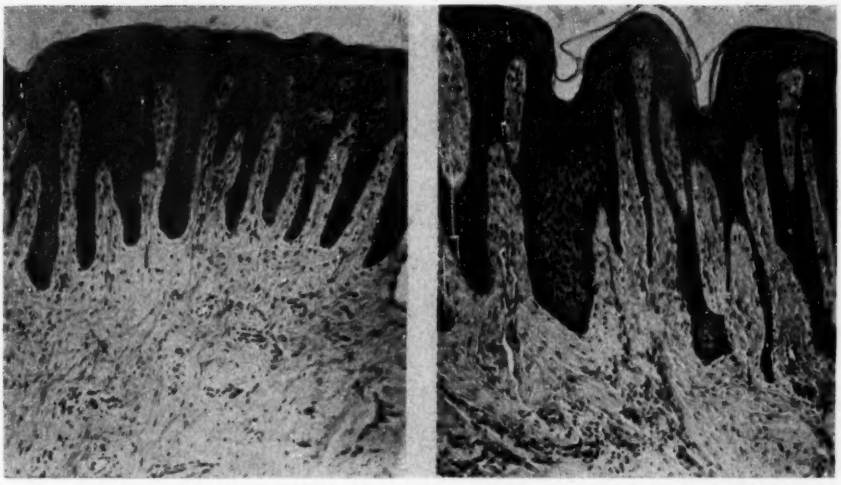
Papillary Layer. A fine textured connective tissue was not noted until the fifteenth year and was equally divided between the sexes. The irregularly textured connective tissue was the most characteristic one in any age group.

The coarsely textured connective tissue appeared for the first time in the 15-29 year group and constituted approximately one-quarter of the specimens.

A sex variation was noted in the 15-29 group. Coarsely textured fibers were found only in male specimens; in contrast, the finely textured connective tissue was found predominantly in female specimens. The variation was so striking that the sex of the specimens could be determined by the histologic section, if the papillary layer of the connective tissue was either coarsely textured or finely textured in this age group.

Reticular Layer. A finely textured connective tissue was found with one exception only in the 4-14 age group. Here, often a mucoid appearing and hematoxylin staining, amorphous, intercellular substance was predominant between the fine fibers of collagen.

The irregularly textured connective tissue was found in more than one-half of the specimens up to 45 years. After that, only 2 of the 25 specimens demonstrated



A. B.

Fig. 5. Photomicrographs of biopsies of a female patient aged 15, (A); and of a male patient aged 16, (B). The 2:2 epithelial histotype of the female contrasts with the 3:3 epithelial histotype of the male. The fine texture of the connective tissue of the former compares with the more coarse texture of the latter.

this arrangement.

A coarsely textured connective tissue was not present before puberty. After puberty to age 45, however, more than one-half of the cases showed this arrangement. Then, in the higher age groups (45-80) approximately all of the specimens showed it.

Connective Tissue Histotypes (Fig. 4). The papillary and reticular layers of the connective tissue varied in character, but similar to the epithelium, formed typical combinations or histotypes. These histotypes varied in some instances in relation to age and sex.

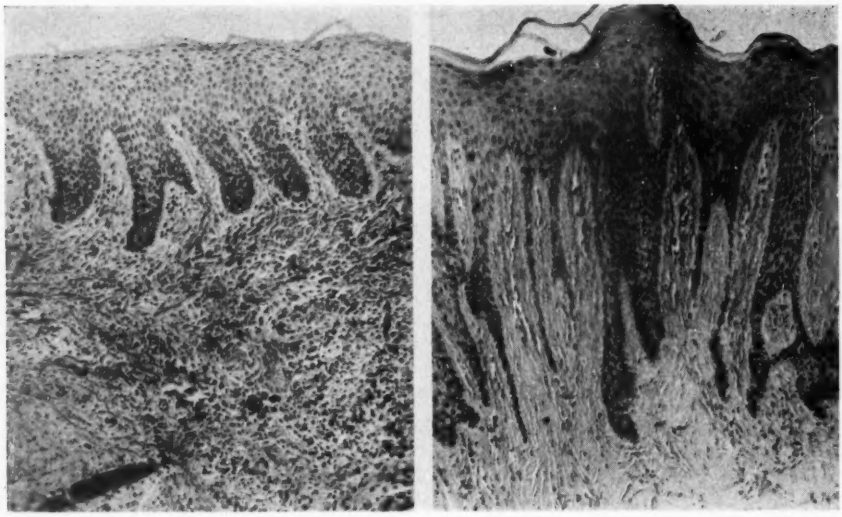
The papillary and reticular layers may be of a finely textured, irregularly textured, or coarsely textured character, designated respectively as 1, 2, or 3. Thus, a 2-3 designation signifies irregularly textured connective tissue in the papillary layer with a coarsely textured connective tissue in the reticular layer.

4-14 Age Group. Three histotypes were found in this group, a 2-1, 2-2, and 2-3. Four specimens were of a 2-1, six of a 2-2, and one of a 2-3 type. The general lack of any coarsely textured connective tissue is significant.

15-29 Age Group. The male group presented a predominant 2-2, 2-3, and 3-3 type (Figs. 4b and 4c), and the female group a 1-2 and 2-2 combination (Figs. 4a and 4b). A trend toward sex differentiation is suggested in the appearance of a 3-3 male type without parallel in the female group and the general tendency of the female to have an overall lower number.

30-44 Age Group. A 2-2, 2-3, and 3-3 histotype was apparent in both sexes. The 2-2 was predominant (14 of 30), followed by the 2-3 group (8 of 30), and finally, the 3-3 (4 of 30).

45-80 Age Group. In this group, there was a 2-2, 2-3, and 3-3 combination. In contrast with the 30-44 age group, different combinations predominate. The 3-3 type was most prevalent (13 of 27) and was found in approximately one-half of the specimens followed by the 2-3 type (11 of



A. B.

Fig. 6. Photomicrographs of biopsies of male patients 5 years of age (A); and 78 years of age (B). Contrast the 2:2 epithelial histotype and the highly cellular connective tissue of the former with the 3:3 epithelial histotype and the sparsely cellular connective tissue of the latter.

27). The 2-2 histotypes in this group appeared in only 2 of 23 specimens.

A trend toward more coarsely textured connective tissue in this group is indicated by the higher numerical index.

DISCUSSION

The constant finding of inflammatory cells near the gingival sulcus is significant, since the specimens were clinically free from inflammation. This microscopic inflammation might be considered as physiologic and to represent a response to a constant bacterial environment in the oral cavity and especially the gingival sulcus.

EPITHELIUM

Hornification. The presence of a well hornified layer in the great majority of the specimens indicates that it is characteristic of the "clinically normal gingiva." Since the patients were institutionalized and suffered varying degrees of mental incompetence, it might be inferred that despite neglect of oral hygiene, the attached gingiva maintained a well hornified surface. The presence of this layer probably played a part in the selection of the specimens as "clinically normal" because the operator selected the tissue on the basis of its pale pink appearance, varying to grayish tinge. Orban[16] states that this appearance is due to the thickness of the stratum corneum and granular layer.

No cytologic change of epithelial cells could be correlated with a part of the aging process. Cowdry[17] states that the epidermis remains young and unchanged by virtue of the basal cells which are vegetative intermitotic cells, that is, their individual life span extends from the mitosis which gives them birth to the succeeding mitoses, when each cell gives rise to two new individuals. The spinous cells are also capable of mitosis and those, whose individual lives end in mitosis, are designated as differentiating intermitotics. Post-mitotic cells are found in the upper layers whose life is terminated in senility and death (hornification) and are lost from the sur-

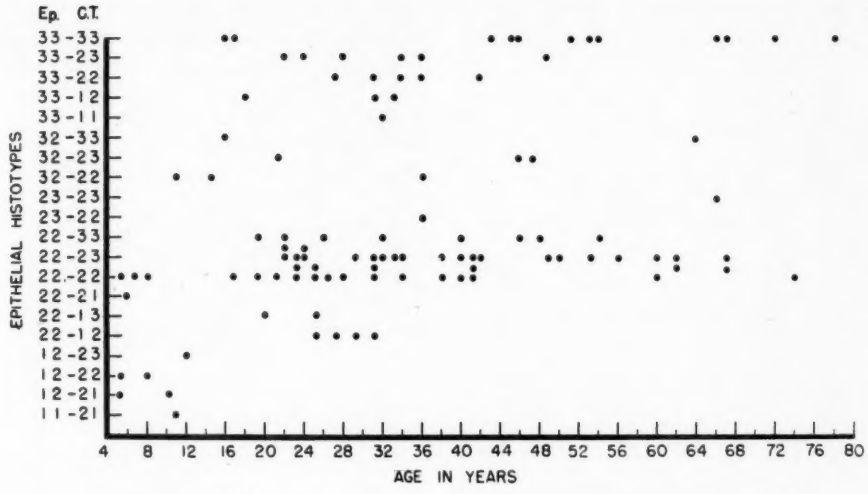


Fig. 7. Diagram showing epithelial histotypes and their distribution according to age. The epithelial histotypes (Ep.) are arranged with the associated connective tissue histotypes (C. T.) of the structure. The distribution shows the 2:2 and 3:3 histotypes are typical for the age groups from 16 to 80 years.

face by shedding.

Epithelial Histotypes (Fig. 3). The arrangement of the cells into various morphologic tissue types or histotypes affords a key to the adaptation of structure to function and to some degree to age.

The finding in the 4-14 age group of a prominent type with short ridges and medium width (1-2 histotypes) indicates an immaturity of these young tissues, since all older specimens showed a longer epithelial ridge length and in some instances a thinner epithelium.

The appearance of the 2-2 and 3-3 types in the 15-29 age group represents an increase in the overall surface area in contact with connective tissue by virtue of the increased epithelial ridge lengths. Thus, the firmness of attachment of epithelium to connective tissue is enhanced and is well adapted to withstand the buffeting of functional activity. The increased contact with connective tissue affords a better contact with the vascular supply of the connective tissue, and, therefore, better nutrition.[18]

Sex Differences. The appearance of a 3-3 histotype, restricted to males, and the characteristic 2-2 designation for females indicates for the 15-29 group a trend toward sex differentiation (Figs. 5a and 5b). The sex differential is lost in the 30-44 and older age groups.

This transient sex difference may be an example of a retarded differentiation in the female, or it may be due to a difference in connective tissue as suggested by Orban.[2]

The 3-3 histotype presented a histologically characteristic wavy outer surface (Fig. 5b), and was correlated to a clinical macroscopic observation of coarse granular stippling of the gingiva. Fine stippling was related to a 2-2 histotype, which in contrast to the 3-3 histotype, might be considered less adapted to mechanical function. The "key" to the interpretation of the various histotypes appears to be a change related to the degree of differentiation and functional maturity of the tissue. The less differentiated, thick, short ridged epithelium of the prepubertal group at puberty

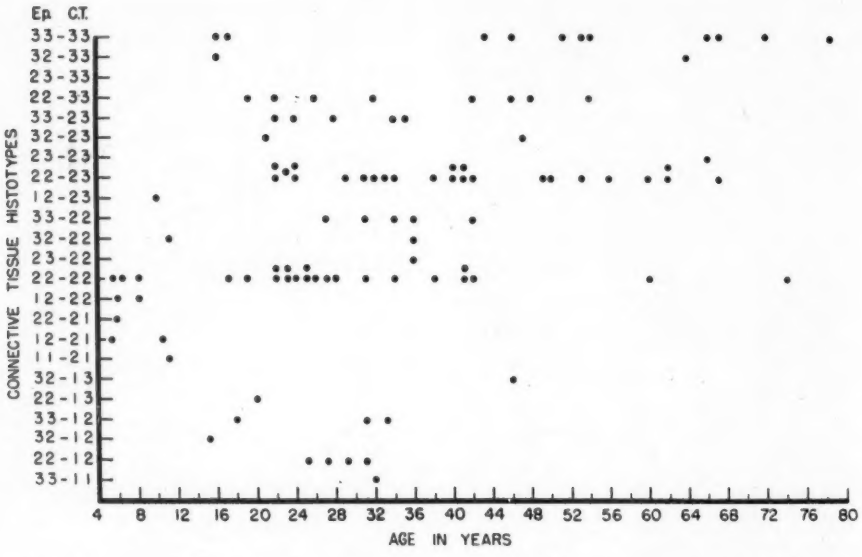


Fig. 8. Diagram showing connective tissue histotypes and their distribution according to age. The connective tissue histotypes (C. T.) are arranged with the associated epithelial histotypes (Ep.) of the structure. The distribution shows a trend toward a higher numbered connective tissue histotype with increasing age, indicating a trend toward a more coarse connective tissue with aging.

develops into the medium thick, medium ridged epithelium (2-2) of the female, while some males show more advanced development into the thin and long ridged type (3-3). After 30 years of age the predominating 2-2, 3-3 histotypes are found in both sexes (Fig. 7).

The histotypes are probably determined genetically.

Sheldon[19] in his studies on constitutional research, states that it may be possible to somatotype cells and to see them in much the same perspective as that in which we are now accustomed to see the human organism as a whole. It may be that "tissue typing" (histotyping), representing as it does a community of individual cells, is a step in this direction.

CONNECTIVE TISSUE

No definite correlation of an epithelial histotype to connective tissue histotype could be made. Epithelial typing was carried on from a quantitative standpoint, that is, width and length; whereas the connective tissue was typed on a qualitative basis —finely textured, irregularly textured, and coarsely textured. In all probability, a quantitative evaluation would reveal the epithelium as a mirror of the connective tissue.

The immaturity of the connective tissue in the 4-14 age group is shown by the loose arrangement of the fibers of the reticular layer (2-1 type) (Fig. 6a).

Sex differential is again suggested by the fine texture of the papillary layer of the female group in 15-29 age (1-2) (Figs. 4a and 5a). The contrasting coarsely textured (3-3) male type was very outstanding in the histologic specimen (Figs. 3c and 5b).

Numerous references to the endocrinologic basis for the female connective tissue

character are suggested. Ingelby[20] states the periductal connective tissue of mammae became more transparent and apparently edematous during the second half of the cycle, due possibly to progesterone. Ziskin[21] indicates that testosterone administered in some gingival conditions causes the formation of more homogenous collagen. Duran-Reynals, Bunting, and Wegenen[22] state that hyaluronic acid and their compounds accumulate in large amounts in certain regions of the skin under estrogenic stimulation, and that it is a reflection of what occurs in the whole skin or whole mesenchyme on a more restricted scale.

Sex differential is lost in older ages. After 45 years, the 3-3 type is predominant, indicating an increased density of the connective tissue in the higher ages.

In summary, the irregular to finely textured pre-pubertal connective tissue shows a gradual transition with age to a more dense coarsely textured tissue in the higher age groups. Seemingly, this progressive change is interrupted after puberty. In this period the female gingiva tends to become more finely textured, while the male shows a trend toward more coarse texture. After 30 years, this sex differential is lost (Fig. 8).

SUMMARY AND CONCLUSIONS

The present study was based on the histologic investigation of the gingiva of 38 female and 64 male patients ranging from 4 to 80 years of age. Each biopsy was an interdental papilla which was clinically free of inflammation and was selected from individuals with a very low index of gingival inflammation. However, microscopically, all specimens showed an infiltration of inflammatory cells near the gingival sulcus.

All findings reported, therefore, will be confined to the attached gingiva because it was not affected by modifying factors of microscopic inflammation.

The findings were as follows:

A. *Architectural Patterns*—Histotypes

1. Epithelium was classified according to two characteristic types (histotypes) of:
   a) Medium suprapapillary width and medium length ridges (histotype 2-2)
   b) Narrow suprapapillary width and long ridges (histotype 3-3)
2. Connective tissue was classified according to its fibrous structure into three types:
   a) Finely textured fibers (rich in cells)
   b) Irregular textured fibers
   c) Coarsely textured fibers (poor cells)

B. *Age Changes.*

1. Epithelial cells showed no significant changes with age. Hornification was characteristic for all ages.
2. The connective tissue showed changes with advancing age.
   a) The cellular elements decreased
   b) The fibrous intercellular substance increased and became coarsely textured.

C. *Sex Changes.* The following sex changes were observed in the epithelium and connective tissue of the 15-29 year age groups:

1. In the females, the epithelium showed a medium suprapapillary width and medium long ridges (histotype 2-2). The papillary layer of the connective tissue was finely textured.
2. In some males, the epithelium showed a narrow suprapapillary width and long ridges (histotype 3-3). The papillary layer of the

lamina propria was coarsely textured.

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ACADEMY SEMINAR

Dr. Edgar James, Chairman of the 1952 Seminar of the American Academy of Periodontology, reports the program is almost complete. The Seminar will be held in Ann Arbor, Michigan, on April 28, 29, and 30, 1952. As usual, the program will include discussions of basic subjects relating to periodontal problems. Details may be obtained from Dr. James, 1802 David Broderick Tower, Detroit 26, Michigan.

Dietary Analysis in Routine Office Practice

by ARVIN WILLIAM MANN, B.S., D.D.S., *Fort Lauderdale, Fla.*
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INTRODUCTION

SYSTEMIC factors are a prime consideration in the diagnosis and treatment of periodontal disease. Although hormonal imbalances, blood dyscrasias, and other systemic factors are extremely important in selected cases, the most routinely noted systemic factor to be considered is nutritional deficiency disease.

As early as 1673, Archer[1] spoke of the great damage which comes to people because they do not know their own "constitution of body; in ignorance, they eat and drink simply to please the palate. Many dig their graves with their teeth and cut off the thread of life sooner than is required by God or nature—besides living in sickness and disease."

While history and clinical examination, laboratory tests and other procedures are of paramount importance in arriving at a diagnosis and planning treatment in nutritional deficiency disease, an analysis of the patient's diet frequently contributes additional information of great value. Unfortunately, dietary analysis is usually employed only in selected cases by most periodontists, whereas it should be a routine investigation in all cases of periodontal disease.

The present report will attempt to evaluate the various methods of dietary analysis at present available in the light of *time* and *effort* allowed the busy periodontist; and the limitations of dietary analysis in the overall picture of the diagnosis and treatment of periodontal disease.

COLLECTION OF DIETARY INFORMATION

Radusch,[2,3] Walsh,[4] and others believe that a seven-day period for collecting dietary information is to be preferred, as a shorter period provides questionable data, while more than one week may be repetitious. However, Jones[5] believes a fourteen-day record gives additional worthwhile information. Care must be exercised not to coach the patient during the period when dietary information is collected, as then he becomes aware of his faults and unconsciously tries to correct them before an evaluation can be made. The patient should write down the *kind* and *amount* of foods eaten as soon as possible after meals to prevent mistakes and omissions. To insure this, the patient is provided with a small notebook in which he can segregate his data under the headings, *breakfast, lunch, dinner* and *between meals*. This seems better than using printed forms, although many dietitians prefer mimeographed forms because they give full instructions on the first page. These are usually standard letter size which do not fit as conveniently into the pocket or purse as does the small notebook. However, if they can be folded into "pocket" size they are probably better, as the writing is frequently illegible in small notebooks.

Another method of obtaining dietary information is to interview the patient, following a standard procedure and recording data in a printed form.* This method is often preferred by dietitians. Trulson[6] states, "Although there is no proof that one method is more reliable than the others —the author prefers the interview of usual food practices, as a method of choice for clinical studies, since it may reveal long-time food practices."

However, these diet records will be of

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*e.g. Form used in some studies at the Department of Nutrition, Harvard School of Public Health, 695 Huntington Avenue, Boston 15, Massachusetts.

A Guide
TO GOOD EATING
MILK
2 OR MORE GLASSES DAILY . . FOR ADULTS
3 to 4 OR MORE GLASSES DAILY . . FOR CHILDREN
To drink, combined with other foods,
in ice cream and in cheese
VEGETABLES
2 OR MORE SERVINGS DAILY OTHER THAN
POTATO . . . 1 green or yellow; "greens" often
FRUITS
2 OR MORE SERVINGS DAILY
At least 1 raw; citrus fruit or tomato daily
EGGS
3 TO 5 A WEEK; 1 DAILY PREFERRED
MEAT, CHEESE, FISH, POULTRY
1 OR MORE SERVINGS DAILY
Dried beans, peas, peanuts occasionally
CEREAL AND BREAD
2 OR MORE SERVINGS DAILY
Whole-grain value or enriched
Added milk improves nutritional values
BUTTER
2 OR MORE TABLESPOONS DAILY
OTHER FOODS TO SATISFY
APPETITE AND COMPLETE
GROWTH AND ACTIVITY NEEDS
Statements on this chart have been accepted by the Council on Foods and Nutrition of the American Medical Association
© Copyright 1943, Revised 1948, National Dairy Council, Chicago 6

Figure 1.

value only if the patient can intelligently speak of the kind and amounts of food and beverage he eats and drinks, if he is motivated to do so, and if the interviewer or history taker has an alert and inquiring mind and accuracy for detail, keen powers of observation, and ability to interview without suggesting answers, sound basic training in nutrition and related sciences, maturity of judgment, and a personality that will gain the confidence and cooperation of the patient.

Unfortunately, few practicing periodontists can devote the time and effort to meet all these qualifications and become competent dietary interviewers. Most dentists are untrained in methods of obtaining correct dietary information by direct questioning and in addition, cannot devote the time required. Inaccurate, incomplete and wrongly interpreted data can produce inaccuracies in the dietary analysis which may contribute wrong and even harmful information to the final diagnosis, and a false feeling of security in the preciseness of the procedure.

METHODS OF DIETARY ANALYSIS

The highest level of dietary analysis is obtained when all food and liquid which the individual eats and drinks is weighed, aliquot parts* of each food and beverage analyzed for the nutrients to be determined, and calculations made from the information using gram percent values as the basis for the calculation of the intake from the dietary consumed. This involves equipment, personnel and procedures which are available only under the most carefully controlled laboratory conditions and is obviously impractical for routine use in the dental office. It is important to note also that this method does not give a picture of the patient's past intake.

The second type of analysis which rates next highest in accuracy includes a record in gram weights of the food and beverage intake and a computation of nutrient content from published tables.* Errors may be introduced by this method as the subject may restrict or change his diet during the test period. Food value tables are only approximate as they are compiled from averages of several laboratory analyses and do not actually represent the actual food values of any one sample. Food values of necessity vary widely when they are obtained from different geographical and climatic sources. The nutrient content changes with different methods of handling, cooking, processing, and etc., so that the food values given in the tables may be quite different from the nutrients actually ingested.

Eakin[7] and Sarett[8] have demonstrated the loss of nutrients in restaurant and institutional foods which produce some inaccuracy in these determinations.

This type of analysis also requires more time and effort than the practicing periodontist can himself give and may not be applicable to routine use in the dental office unless trained auxiliary personnel is available.

Other methods of calculation of food values have also been advanced. Leichsenring, et al.[9] have introduced a shorter method of analysis by segregating food into groups before calculation of values.

Miller[10] and Blass[11] also segregate food into groups by utilizing a form containing lists of foods in food groups. Squares are aligned opposite each food and caloric value assigned to each. Entries from the

*An analysis of aliquot parts is only one of several methods possible.

*(a) Composition of Foods, U.S. Dept. Agr. Handbook No. 8, 1950.

(b) Food Composition Tables for International Use. Food and Agr. Organization of the United Nations. October 1949.

(c) Sodium and Potassium Analysis of Foods and Waters. Mead, Johnson & Co., October, 1941.

(d) Food Values of Portions Commonly Used. Bowes, A. & Church, C. P., Philadelphia, Pa., 1951.

(e) Food and Beverage Analysis. Bridges, M. A. & Mattice, M. R., Lea & Febiger, Philadelphia, Pa., 1942.

patient's seven-day dietary record are checked off, one to each square. The values are then totalled and divided by seven. The dietary caloric and nutrient intake is calculated and compared with a normal dietary requirement table* to determine specific dietary inadequacies. Specific recommendations for dietary improvement are then made to the patient.

Jones[5] uses a method of dietary analysis in a practice limited to diseases of the eye, ear, nose and throat, which may easily be applied in routine periodontal practice. Instead of attempting to quantitatively measure amounts and kinds of food eaten, *dietary habits* are studied to determine *good* and *bad* dietary habits, by classifying the foods in a fourteen-day dietary into *good* and *bad* categories. A large pad of legal sized paper, separated into two sections by a vertical line down the center of the page, is then divided into four sections horizontally and the sections labelled, *breakfast, lunch, dinner* and *between meals*. The entire left column is labelled *good*, and the right, *bad*. The patient *reads* his own entries off and the dentist judges which category the food is to be entered into and makes notes in the section allotted. The *pattern of dietary habits* will thus unfold and an amazing amount of dietary information will be obtained by this very simple method. The notes are then studied and specific recommendations made to the patient to change his bad habits. The patient should be complimented on his *good* habits when the dietary is discussed, as this will encourage him to improve his diet by eliminating his *bad* habits. This method naturally cannot be compared for routine accuracy with the more objective quantitative methods, but it does yield very valuable information which is usually missed, and has the great advantage over all other methods available in that it requires little time and effort and can be carried out routinely as a part of every periodontal diagnosis.

Obviously, a knowledge of *good* (Fig. 1 reprinted by special permission) and *bad* foods and *good* and *bad* dietary habits is a prerequisite to the use of this method. The more important habits can be mentioned. Omitting breakfast is a common, glaring dietary fault. Routine analysis by this method in one of the author's (AWM) practices reveals that only one-third of patients interviewed eat an adequate breakfast; one-third eat a definitely inadequate breakfast; and the remaining one-third *do not eat any breakfast at all*. Since breakfast should suppy its share of the total daily dietary requirements—the patients in the last two groups obviously start off the day being deficient. The old fashioned coal furnace furnishes us a very good example of good dietary habits. We should open the draft and really pile on the coal in order to get the house warm in the morning (breakfast); adding only enough fuel to keep the house warm during the day (lunch) and evening (dinner); and checking and banking the furnace to conserve fuel during the night. Many of our periodontal patients unfortunately reverse this procedure by omitting breakfast, skimping lunch, and eating a huge dinner at night.

Another common dietary fault is to attempt to compensate for dietary inadequacy during the day by drinking a mid-morning and mid-afternoon carbonated beverage. This may give the patient a "lift" in calories, but certainly does not supply the nutrients required. The substitution of refined carbohydrates for protein and fat in the diet and failure to incude foods supplying the trace minerals, (e.g. seafoods) in the dietary are of prime interest to the periodontist because long-standing deficiencies of these nutrients help to promote periodontal disease. Laing[12] has ably discussed the importance of these nutrients in gaining and maintaining health. A multitude of other bad habits help to produce nutritional deficiency disease by making for inadequate dietary intake every day.

*e.g. Recommended Dietary Allowances — National Research Council Reprint and Circular Series No. 129 (October) 1948.

LIMITATIONS OF DIETARY ANALYSIS IN THE OVERALL DIAGNOSIS AND TREATMENT OF PERIODONTAL DISEASE

Periodontists vary widely in their beliefs as to the relative importance of systemic and local factors in the diagnosis and treatment of periodontal disease. If systemic factors are to be considered, certainly an analysis of the patient's dietary should be attempted in addition to obtaining clinical and laboratory information, if we are to obtain a more thorough diagnosis of nutritional deficiency disease. It should be emphasized, however, that *diagnosis of nutritional deficiency disease should never be made or treatment planned on the basis of dietary analysis alone.* Only by combining this information with *clinical and laboratory data* will specific nutritional deficiency syndromes be identified and intelligent treatment planned.

DISCUSSION

If the periodontist is so fortunate as to have the services of a trained dietitian readily available, the methods first described in this paper are by all means to be recommended. If, however, he is burdened with the routine of office practice without trained dietetic assistance, the method outlined by Jones[5] works very well although it makes no attempt to be quantitatively accurate. It has the decided advantage in that it can be used with a minimum of time and effort in every periodontal case. The authors believe that accurate dietary information will be a help in the diagnosis and treatment planning of periodontal problems and urge you to make dietary analysis a routine procedure in your offices, using the method which best suits your individual circumstances.

SUMMARY AND CONCLUSIONS

1. The importance of dietary analysis in the overall diagnosis and treatment of periodontal disease has been stressed.

2. The methods of collecting dietary information were described.

3. The different methods for analyzing the dietary were outlined and the advantages, disadvantages and limitations of each discussed.

4. A method of analysis requiring a minimum of time and effort which can be used routinely in all periodontal cases was recommended, where time, effort and personnel limitations prevent using conventional methods of dietary analysis.

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Gingivectomy—Its Place In Periodontal Therapy*

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INDICATIONS:

GINGIVECTOMY is the surgical removal of excessive free gingival tissues in order to enable the patient to keep his teeth clean. When following instructions for home care, after a gingivectomy, the patient should be able to reach all exposed surfaces of his teeth. Besides attaining elimination of periodontal pockets, gingivectomy also may be utilized as a gingivoplastic procedure in cases of hyperplastic gingivitis.[8] Following a subacute or chronic necrotizing ulcerative gingivitis,[2] the loss of tissue often has created an interproximal crater bounded by buccal and lingual gingival walls which prohibit an effective oral hygiene. In such cases a gingivoplastic procedure which often includes a bone plastic, may be indicated to give the patient access to the interproximal areas.

In order to discuss the indications for gingivectomy, Orban's[24] classification of periodontal pockets is used:

1. The gingival pocket, pseudo-pocket, or false pocket, is found in gingivitis. The gingival crevice is deeper than normal, due to edema and inflammatory swelling of the free gingiva. A slight down growth of the epithelial attachment may have occurred but no bone resorption is found.

2. The periodontal pocket, is a pathologically deepened gingival crevice characterized by a displacement of the epithelial attachment in an apical direction along with bone resorption. The bottom of the pocket is situated coronally to the alveolar crest.

3. In the intra-alveolar, intraosseous, or intrabony pocket the bottom of the pocket extends apically to the margin of the alveolar crest. With pocket formation, irritation and inflammation always can be observed in the gingival tissues and most periodontists feel that a gingival crevice deeper than two to three mm. can not be kept clean. Retention of food products in a gingival crevice will act as a source of irritation and periodontal inflammation will cause continued extention of the pocket. This deep crevice, therefore, constitutes a hazard to the maintenance of the tooth.

Gingival pockets of Type 1 will disappear in most cases following a thorough oral prophylaxis, which should include: (1) removal of all calcerous deposists, plaques, overhanging restorations, (2) smoothing and polishing of all exposed tooth surfaces and rough restorations, (3) equilibration of occlusion, (4) correction of faulty proximal contacts, (5) reestablishing of tooth contours, (6) repair of all carious teeth, (7) replacement of lost teeth, and (8) good home care, including tooth brushing and gingival stimulation. In some cases of gingival hyperplasia the oral prophylaxis has to be followed by a gingivectomy or a gingivoplastic procedure in order to eliminate the deepened crevice. Only the free hyperplastic gingiva above the bottom of the epithelial attachment should be removed. The excision should conform to and facilitate the formation of a proper gingival contour. Gingivectomy, as an initial or early treatment for gingivitis is contraindicated for the two following reasons:

1. Infected and inflamed tissues should not be operated if surgery can be avoided.

2. In most cases of gingivitis, there are remaining free gingival fibers present (Figure 1). These fibers will, when the source of irritation is removed and the inflammation has subsided, facilitate the restoration of a normal free gingiva, which is the goal of the treatment.

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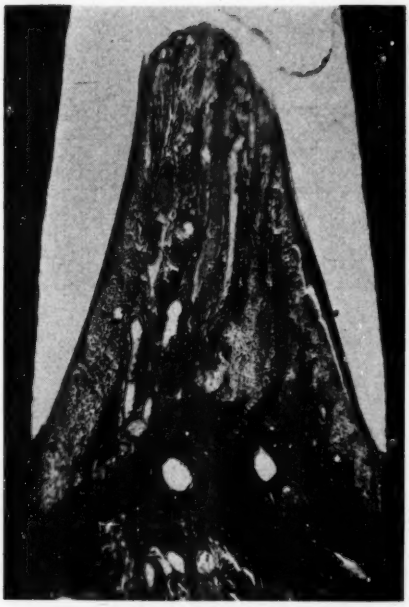

Fig. 1. Azan stain. Residual free gingival fibers in a case of gingivitis

Occasionally the oral prophylaxis, even in presumptively healthy persons, does not produce the expected good response and the dentist may be tempted to perform a gingivectomy in order, "to try everything." The threshold for the amount of irritation required to produce inflammation is so low in those individuals that the local inflammation is maintained by negligible irritants which are easily overlooked by the dentist. Cases of that type often have been listed without proved justification, as cases of systemic or endogenous gingivitis. Then, the only chance for cure or control of the disease is by improvement of local therapy and the patient's home care, and these cases should constitute a special challenge to the operator for thoroughness in his periodontal procedure. A "trial gingivectomy" will act as an additional irritation to the gingival tissues when resistance is poor, and the low potentiality for healing in such a patient will create a gingivitis following the gingivectomy which will be even worse than before the operation was performed.

Pockets of Type II, the periodontal pockets, are commonly found in association with periodontal disease. A large percentage of these pockets also will disappear during the initial hygienic phase of the periodontal treatment.[6] The free gingiva, constituting the soft tissue wall of the pocket, is edematous, hyperemic, inflamed and swollen. Initial removal of the local irritants by oral prophylaxis and occlusal equilibration, followed by massage of the gingiva, will improve the circulation and the shrinkage of the gingiva during the process of the healing often will eliminate the pocket. But in some cases in which the pockets are deep and chronic proliferative inflammation has led to fibrosis of the free gingiva, the pockets may persist. These residual pockets can be eliminated in two ways:

1. By subgingival curettage leading to periodontal reattachment.

2. By gingivectomy.

The theoretical and practical possibility of periodontal reattachment has been debated widely during the last fifty years. Clinical and experimental investigations have shown that periodontal reattachment only can be obtained if the local inflammation can be brought down to a minimum before subgingival curettage is attempted to secure reattachment.[27] Horizontal loss of bone and numerous deep pockets in the premolar and molar region makes it difficult to remove the calculus properly, and makes it almost impossible for the patient to keep those pockets sufficiently clean for the inflammation to subside and for the gingival tissues to attain a state of health which would make rational an attempted reattachment. Consequently, gingivectomy is the most practical choice for elimination of residual pockets, deeper than 2-3 mm. in the bicuspid and molar region. Access to the incisor and bicuspid regions is much easier than to the molar regions both for the operator and for the patient. Therefore, the possibility for reattachment following

subgingival curettage is best in the anterior region of the mouth. The esthetically unsatisfactory results following a gingivectomy in this area should also encourage a conservative approach. Conservative treatment may be more time consuming than surgery and only a relatively small group of dentists may have acquired sufficient technical skill in subgingival curettage to obtain a good result.

Since the dentist's responsibility is to provide the best known health service to his patients, arguments against conservative treatment are invalid if they are based on inconvenience in utilizing a hitherto neglected technic. A similar problem has been faced in endodontia. The old fashioned root canal therapy of immediate insertion of a paste filling without proper control, required little time and was easy to perform, but it resulted in a failure which was followed by an equally poorly planned apicoectomy, as the next step toward the loss of the tooth. The satisfactory root canal treatment of today, is much more difficult and time consuming than the old therapy, but this improvement of an old procedure in dentistry has led to a much lower percentage of root resections and loss of teeth than a couple of decades ago. It is the feeling among many periodontists that the indications for gingivectomy likewise could be narrowed considerably by improving one's conservative treatment and by subgingival curettage of the periodontal and intra-alveolar pockets.

In treatment of the third group, the intra-alveolar pockets, a subgingival curettage always should be attempted. This conclusion appears valid, in the first place, because the conservative treatment in these instances offers a good chance for reattachment, and, secondly, because a gingivectomy in these cases eliminates functionally valuable supporting tissues both for the tooth involved and for the adjacent teeth (Fig. 2). A single deep intra-alveolar pocket which cannot be successfully treated by conservative treatment often is eliminated by the extraction of the tooth rather than sacrificing good periodontal support of the adjacent teeth. Each case requires individual consideration. A "spot gingivectomy" to eliminate a pocket of this type will be a failure because the deep dip in the free gingival contour, made in order to eliminate a single deep pocket or an isolated interproximal area with deep pockets, always will fill in again with connective tissue and the pockets will reappear. A single buccal or lingual pocket may in some cases, be permanently eliminated by a localized gingivectomy. The gingival "flap operation" is a radical form of subgingival curettage which will not be discussed in this paper.

Fig. 2. Mandibular first molar in traumatic occlusion. Deep mesial intra-alveolar pocket

II. CONTRAINDICATIONS

The performance of a gingivectomy is contraindicated in an area which the patient cannot keep clean in the future. Pockets extending to the trifurcation area of the maxillary molars represent such a hopeless situation. The exposed trifurcation cannot be kept clean by the patient and subsequently periodontal inflammation will cause a progressive loss of supporting structures. Only in exceptional cases with widely separated roots and a deep muco-buccal fold can a mandibular molar with an exposed bifurcation be kept clean following a gingivectomy, so, in most cases, an exposed bifurcation constitutes a contraindication for gingivectomy.

The contraindications for gingivectomy may be summarized:

1. Acutely inflamed gingiva.

2. Exposed trifurcation and bifurcation of the molar teeth. (In most cases.)

3. A caries attack rate so high that future maintenance of the teeth is impractical or impossible.

4. Esthetically unsatisfactory results anticipated from the operation.

5. Teeth in hopeless mal-position.

6. Seriously lowered general resistance, (certain blood and heart diseases, nephropathies or severe nutritional deficiencies).

7. Patients who in the initial hygienic phase of the periodontal treatment, did not cooperate sufficiently to be reliable about their home care.

Fig. 3. The alveolar crest is lost from periodontitis, and the inflammatory reaction extends into the adjacent marrow space. Residual spiculae of alveolar bone maintaining functional relation to the teeth.

THE EXTENT OF TISSUE REMOVAL IN GINGIVECTOMY

Knowledge of periodontal histology, physiology and pathology has increased considerably during the last two or three decades, making a reevaluation of the gingivectomy procedures desirable. The radical surgery recommended by Widman[35] and Neumann[18] and later somewhat modified by Ward,[32] was based on the erroneous assumption that pyorrhea alveolaris primarily started out as a disease of the alveolar and supporting bone.[1,19] Therefore, in the old literature much emphasis was placed on removal of what was called "the decalcified, diseased and necrotic bone,"[36] then considered an obstacle to the healing process. The bone changes encountered in periodontitis and periodontosis are now considered to be secondary to pathologic processes in the soft tissues surrounding the bone.[15,34] Bone resorption is caused by osteoclastic activity and the osteoclasts are cells differentiated from the soft tissues surrounding the bones. When the surface irritants which were responsible for the inflammatory process are removed and dysfunctional forces eliminated, the osteoclastic activity will cease, and functional stimulation of the periodontium will promote healing.

It is important to maintain the remaining marginal spiculae of the alveolar bone because they are often in functional connection with the tooth and will facilitate a regeneration of the alveolar crest to its highest position[20] (Fig. 3). One should not expect any appreciable regeneration of bone above the marginal level of the remaining bony spiculae or above the level to which the bone has been removed during the surgical procedure. There is no histological or practical rationale for the curettage of the alveolar crest, removal of residual bone and the mutilation of the periodontal tissues which sometimes is recommended.

Another common fallacy is the emphasis which is placed on removal of all granulation tissue[3,4,12,13] in the area of the gingivectomy. Granulation tissue is commonly seen below residual periodontal fibers and

the peri-vascular spread of inflammation in periodontal disease[34] will often cause formation of granulation tissue extending deeply into the adjacent marrow spaces, even below the alveolar margin (Fig. 4). This tissue is produced as part of a tissue defense reaction and it represents the first attempt of healing in an area of previous destruction. If the irritants which cause and maintain the inflammation can be eliminated, the granulation tissue will participate in the process of healing within the biological possibilities for repair which the injured tissues possess. A removal of granulation tissue below the bottom level of the epithelial attachment would necessarily involve removal of valuable periodontal fibers fastened both to the tooth and the bone, and it should be obvious that one's treatment would end with the epithelial attachment more apically situated than before the operation (Fig. 5). This simple but fundamental fact is commonly overlooked even by the most recent writers[13] who recommend removal of the gingival tissues to the existing level of the alveolar bone.

There has been some discussion about the attachment of new fibers in an area of gingivectomy.[16] The postoperative surgical packs will allow for only a thin blood clot on top of the cut surface. This clot may be replaced by granulation tissue and differentiated into cementoblasts and periodontal fibers which may prevent the ingrowing epithelium from reaching the demarcation line of the operation. Even the most favorable conditions, such as a high potentiality for healing and a minimum of inflammation during the period of healing, can not prevent some ingrowth of epithelium into the organizing bloodclot.[10, 25] Later some regeneration of the interdental connective tissue will raise the level of the interdental area but the epithelium never will attach itself to the tooth surface above the initial epithelial attachment which was established soon after the operation (Fig. 6). The rationale for a gingivectomy is not, as stated commonly in the literature to remove diseased tissues, but the operation is performed in order to remove a sufficient

Fig. 4. Severe periodontitis with granulation tissue extending below residual transeptal fibers and apically to the alveolar crest.

amount of the soft tissue wall of the pocket to allow access for future home care and thereby prevent irritation and periodontal inflammation. Severe gingival infection with accumulation of bacteria on the gingival surface and toxic products accumulated in the underlying pyogenic granulation tissue would interfere with the process of healing. But, as previously stated, a gingivectomy should never be performed in this active stage of the periodontal disease. Removal of the irritants and later stimulation of the circulation by massage will eliminate the toxic products in the gingival periodontal tissues. Consequently, if a proper mouth preparation precedes the gingivectomy, the problem of infected, inflamed granulation tissue will not exist.

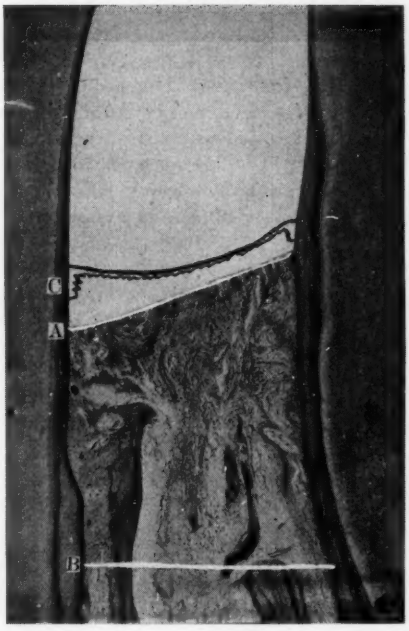

Fig. 5. The same case as shown in Fig. 3. The removal of gingival tissues should not extend below the line at "A." The white line at "B" indicates the extent of a radical gingivectomy (Widman, Neumann, Ward). A surgical pack will protect the organizing blood clot on the surgerized area until regenerating epithelium has covered the surface and established a new epithelial attachement ("C").

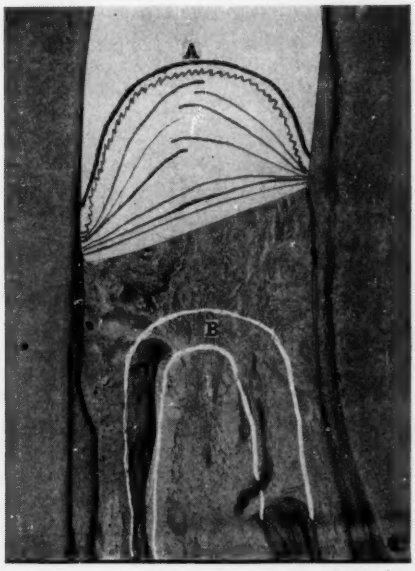

Fig. 6. This illustration shows attempt to outline what would constitute a good result of a gingivectomy in the case shown in Figs. 3 and 5. The line at "A" indicates the surface of the new interdental papilla. Newly formed transeptal and gingival fibers are indicated. The alveolar bone and alveolar crest should regenerate as outlined in white ink ("B").

GINGIVECTOMY PROCEDURE

An evaluation of the different technics which have been recommended for gingivectomy is only possible if one has a clear concept of why a gingivectomy should be performed and to what extent the gingival tissues should be removed. Exact control of the elimination of tissue during gingivectomy is of utmost importance in order to assure the removal of the least required amount. Residual pockets should be eliminated and proper gingival contour secured without unnecessarily denuding previously unexposed tooth surface. Therefore, no technic involving radical gingival surgery should be used. The extent of the effect of caustic packs[9, 22] and electrocoagulation[5, 26] cannot be controlled accurately. The use of these technics should, therefore, be restricted to the very few cases in which local anesthesia can not be administered. Different sets of steel knives have been designed for gingivectomy. The choice of instruments is of minor importance depending mainly on personal experience and acquired skill. Lately, high frequency ultrashort current has been extensively used for minor surgery,[7, 11, 14, 28, 29, 30, 33] including gingivectomy. Correctly used, an electrosurgical scalpel is easy to handle and permits good results. The cutting should be performed with the lowest amount of current that will cut the particular tissues without appreciable resistance. A steady, fast cutting motion will result in a very shallow surface destruction, less than 0.1 mm. in depth.[31] A smooth pulp canal broach can be used as an electrode and this instrument easily can be adapted for use in

the interdental spaces. The hemorrhage is less than that encountered following the use of steel knives, the contouring of the gingiva is facilitated and an additional cut easily can be made if too little tissue were removed in the initial incision. Sealing of the capillaries and the lymphatics also prevents spread of infection into surrounding tissues.[11, 14]

The electrosurgical scalpel is, because of the ease by which it cuts soft tissues, a dangerous instrument in the hands of a non-experienced operator, and there are certain objections to its use. The smell of burned flesh accompanying the cutting is disagreeable to patients, and the conduction of electric current through large fixed bridgework also is unpleasant. The cutting needle should never be brought in contact with the periosteum or bone[17] and it should not be moved slowly in close proximity to the periosteum. If bone has to be removed in order to obtain a proper gingival contour that should be accomplished by the use of either a bur, a bone file or a chisel and the exposed bone should be covered by a blood clot before the postoperative pack is placed.

Packing of the pockets is recommended before the gingivectomy. Small, about an inch long, loosely rolled cotton spindles or strings, soaked in Ward's Wonder-Pack (or another surgical cement) mixed to consistence of thick cream should be placed carefully in the pockets. These packs should be left in the pockets for one week, and if they have been placed correctly a good view of the pockets is obtained when the packs are removed. This procedure helps the operator to avoid removal of tissues apically to the bottom of the epithelial attachment. The epithelial attachment in the bottom of an untreated pocket is usually short (See Fig. 3) so the surgical intervention should extend only slightly (0.5-1 mm.) below the bottom of the pocket. As previously indicated in Figure 5, a very short distance of reattachment might in a few cases be obtained following a gingivectomy, therefore the epithelial attachment should be removed throughout its entire extent and an effort should be made to assure a good blood clot on the wound contacting the tooth surfaces before the postoperative surgical dressing is applied. The first dressing should be removed four to five days following the operation and the surface of the wound rinsed cautiously with a one per cent lukewarm solution of hydrogen peroxide and a new dressing should be placed over the area. The cut surface always will accumulate some slough, formed by traumatized coagulated tissue particles, fibrin, migrated and necrotized leukocytes, and the slough will, under the influence of saliva and bacteria from the mouth seeping under the pack, start a proteolytic fermentation with the production of toxin, which will disturb the healing process and stimulate proliferation of excessive granulation tissue. The second pack should be removed ten to fourteen days after the operation and at which time the wound surface usually will be epithelialized. If not, the pack should be replaced for another five or six days.

There are four main causes for the formation of excessive granulation tissue in a gingivectomized area:

1. The gingivectomy was performed on severely inflamed gingiva without sufficient preceding oral prophylaxis, and the toxic products present in such an area of active inflammation will produce excessive granulation tissue.

2. The tissues were traumatized by poor surgical technic leaving a ragged cut surface.

3. Loss of the surgical pack or removal of the pack before the cut surface was epithelialized. An unepithelialized and uncovered wound surface naturally will permit an outgrowth of granulation tissue.

4. The first surgical pack was left in the mouth too long allowing the underlying debris to stimulate the adjacent tissues to produce excessive granulation tissue.

If excessive granulation tissue is present, following removal of the pack the tissue should be removed cautiously, by a caustic

but without damage to the surrounding tissues. The caustic which is used should be prevented from floating out on surrounding areas, but, following a properly planned and skillfully performed gingivectomy, there should be no excessive granulation tissue and consequently no need for caustic drugs. There is no rationale for the general use of mild caustics on the healing epithelial surface of the gingiva as recommended by some writers.[21, 23]

Bare bone following uncautious use of the electrosurgical scalpel is an unpleasant complication. It is painful for the patient and it requires several changes of dressing until the denuded bone has undergone sequestration. The pathologic process is basically the same as in a case of "dry socket." The last, but not least important phase of the gingivectomy, is to teach the patient the proper home care and to make sure that he is maintaining that home care. This part of the treatment actually may require more time and be more of a strain, both for the patient and the dentist than the surgical procedure itself. But the successful outcome of the gingivectomy is entirely dependent upon adequate postoperative care and the patient's cooperation.

SUMMARY

1. Gingivectomy is indicated for the elimination of gingival and periodontal pockets deeper than 2-3 mm., when the pockets cannot be eliminated by conservative treatment and when the patient can keep the exposed surfaces of his teeth clean, following the operation.

2. Gingivectomy should be performed only following a thorough oral prophylaxis and for patients who show a willingness and ability to maintain good oral hygiene.

3. The least possible amount of periodontal tissues which is required for elimination of the pockets and securing a proper gingival contour should be removed.

4. Responsibility for the adequate teaching and control of the patient's postoperative home care must be assumed by the dentist. It is part of the treatment and a major feature in achieving a good result.

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A Concept of Occlusion in Periodontal Practice

by BALINT ORBAN, M.D., D.D.S.,* *Colorado Springs, Colorado*

THE question of diagnosis and plan of treatment of an occlusal disharmony was long obscured by the fact that no clear understanding had been reached as to what constituted a "normal" occlusion. However, some progress has been made in the right direction because we are now viewing the problem of occlusion more and more from a functional, rather than from a static point of view. For some time, we were taught that a normal occlusion is one in which the intercuspation of the first molars occurs in such a manner that the mesio-buccal cusp of the upper first molar meets with the groove between the mesial and distal cusps of the lower first molar. This definition of normal occlusion is a static one, and is therefore unsatisfactory from a functional point of view.

Prosthodontists look upon occlusion from a more functional viewpoint and define the occlusal position as that with maximum intercuspation of teeth. Maximum intercuspation, to be considered normal, should coincide with centric position. Centric relation is defined as that position of the mandible in which the condyles are in the most retruded, but *unstrained* (Sicher), position in the glenoid fossae, from which lateral movements are possible. We come to the realization that occlusal and centric positions can be the same, or they may differ. If they coincide, we have a normal occlusal relation and if they do not coincide, the occlusion is not normal. From the viewpoint of a prostho-

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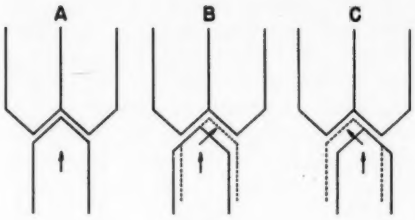


Figure 1

Three different relations of centric and occlusal positions.

A—Centric position and occlusal position are identical. The patient closes from rest position into occlusion without interference.

B—The mandible moving from rest position into occlusion, meets interference and glides anteriorly into occlusal position. (Anterior displacement).

C—The interference is anteriorly and the mandible shifts distally into occlusal position (Distal displacement). In 100 successively examined patients, no such case was found.

dontist, this strict interpretation of a normal occlusion is of great importance. No one would think of constructing a full denture out of centric, and thus not in normal occlusion. How does this principle hold true if we deal with a natural dentition? Examination of 100 individuals, about one-third of whom were dentists, revealed that in 60, the occlusal position was anterior to the centric position; that is, the mandible was in an anterior displacement when in occlusion. Graphically, this position is illustrated in Figure 1-B. Two of these individuals also showed a lateral shift in addition to the anterior one. Forty individuals showed "normal" occlusal relations; that is, there was a harmony between centric position and occlusal position,—(A in Figure 1). One individual had a lateral shift. No individuals were in distal displacement when in occlusion (C in Figure 1).

If one stops to analyze such statistical data, one immediately has to realize that the prosthodontists' "normal" might not be normal for natural dentitions. One might say that the prosthodontists' "normal" constitutes an ideal which, in the natural dentition, occurs only in less than half the cases. If one realizes all the intricate mechanisms of tooth development, eruption, shedding, occlusal attrition, wear on contact points, etc., which have to occur in harmony to end in an ideal occlusal relation, one cannot help being puzzled that the ideal *ever* occurs. Dentists who deal with the natural dentition have to accept at the present, that it is ideal when occlusal position and centric position coincide to give normal occlusal relation, but that what we call "normal" has slight variations. Within certain limits, supporting tissues are built in a manner to adapt themselves to variations in the functional requirements in a rather wide range. This functional adaptation is a basic physiologic reaction of our tissues, without which the human species would be extinct. The supporting tissues of the teeth; the gingiva, the periodontal membrane, the cementum, the alveolar bone, have wide ranges of adaptability to functional variations.

This concept does not mean that we should neglect disharmonious occlusal relations in a natural dentition, as something within the normal range of variations. If such a disharmony exists in a dentition otherwise healthy, with good supporting tissues, without causing any subjective or objective symptoms, there may be no need to interfere with this occlusal relation. However, if the individual's systemic condition brings about a lowered resistance, or if the supporting tissues are diseased, as they are in the various types of periodontal diseases, we should aim to eliminate the disharmony between the centric and occlusal positions to prevent damage to the supporting tissues by secondary traumatism. We have to create the most favorable and least damaging functional relation between the opposing teeth.

In a dentition with a healthy supporting tissue apparatus, the variations from ideal will be met by proper functional tissue adaptation, but in a dentition with diseased supporting tissue apparatus, the mechanism of functional adaptation is disturbed and the range of the adaptability

becomes narrow.

Figuratively, we can compare "normal" with a broad highway, with a line in the center indicating "ideal." With a good functional adaptability of our supporting tissues, we can travel this broad highway safely, deviating from the center to the left or to the right. If, however, the adaptability of our tissues has been reduced due to disease, then the deviation from ideal has to be narrowed. We have to stay as close to the center line (ideal) as possible.

Correction of a disharmony between centric and occlusal positions is only one phase in the correction of occlusal relations. Interferences in protrusive and lateral movements within the functional range, and sometimes even beyond, have to be eliminated or reduced. In several of the cases in group A—Figure 1, other disharmonies existed; deep overbite occurred, as well as other malocclusions.

It is not the purpose of this paper to go into detail regarding diagnostic and corrective measures; only basic principles have been discussed! No relation, whatsoever, could be established between the presence of an occlusal disharmony and the periodontal status.

SUMMARY

The rationale for the correction of an occlusal disharmony is the prevention of secondary damage to the supporting tissues by traumatism. In the various forms of periodontal disturbances, the supporting tissues of the teeth are diseased and their adaptability to functional changes is reduced. We should strive to create the most favorable functional relation possible between the opposing teeth, by eliminating disharmonies between centric and occlusal positions, as well as in eccentric movements.

DR. CARLOS H. SCHOTT HONORED

Dr. Carlos Henry Schott, D.D.S., F.A.C.D., F.A.A.P., was honored at a delightful banquet of the Cincinnati Dental Society in Cincinnati on November 19, 1951. As Dr. Eddie Ball said, "The party was tremendous" with friends of Dr. Schott coming from all over the Midwest to pay their respects. He also received hundreds of letters and telegrams.

It would be difficult to find another man in the American Academy of Periodontology worthy of such an honor. In the many years Dr. Schott has served the Academy, he has gained the respect and admiration of everyone. He was President of the Academy in 1926, elected to Fellowship in 1931, and has been active in many ways year in and year out.

We know that the members of the Academy join the Editor in extending to Dr. Schott congratulations and sincere good wishes. We wish to comment also that the Cincinnati Dental Society is to be congratulated for organizing this fine party; in fact, the Society honored itself by honoring Dr. Schott.

The Cemento-Enamel Junction

by A. B. RIFFLE, D.D.S., *Rochester, N. Y.*

THE cemento-enamel junction is a landmark of particular importance to the periodontist. It is the line of demarcation between the normally hard, highly resistant enamel and the comparatively soft root surface. It marks the incisal/occlusal line of attachment of the periodontal ligament in the very young, and on a few fortunate individuals in late life.[1] When exposed, it becomes the site of cervical hypersensitiveness, cervical caries, and cervical abrasions, all bugaboos to the periodontist and the general practitioner alike.

Microscopically, the normal has been well described;[2] pathologically, references to the cemento-enamel junction as an entity are rare—most studies being of the cementum itself, not the junction; periodontally, there are many clinical observations but few definite studies, most writers treating the area as being of little significance.[8, 27] Nobody has described the physical deteriorations about to be demonstrated.

For example, Miller[3] recognized it as the original site of cervical abrasions, and Hirschfeld[4] pointed out that it is the location of the deepest penetration in these acquired defects. Oulliber[5] noted that teeth long exposed nearly always show more or less etching at the cemento-enamel junction. Gottlieb[6] and Weaver[7] believe that cervical decay has already started at the cemento-enamel junction before the gums recede. Hartzell[8] and Norris[9] recognize sharp spicules of enamel at the gingivo-enamel junction. Noel[10] noted these softenings; that they were more frequent on the labial and buccal surfaces of incisors, canines, and bicuspids; more in the upper than the lower; that they simulated erosion, with a sharply defined edge of enamel; and recommended their removal by grinding and reshaping the area so as to leave it hard, smooth, and easy to keep clean.

It is difficult to discuss the cemento-enamel junction without entering a discussion of the cementum itself—a project reserved for later publication. Sufficient for this article is to state that most present day teaching treats this area: *first*, as if the cementum were necessarily there; *second*, as if, when present, exposed cementum which has lost its soft tissue attachment is sufficiently hard and resistant that it will remain a permanent or even a reasonably long-lived protective covering for the root surface; and, *third*, as if a normal, recently exposed cemento-enamel junction could be curetted with a sharp instrument to a hard, smooth surface without removing the cementum in its entirety.

In regard to the first: In 10% of teeth there are various aberrations in the relationship of the cementum to the enamel, many of which leave an exposed zone of dentin, and in 30% the cementum meets the enamel in a sharp line, which line of junction is even thinner than the cementum itself—already as thin as a human scalp hair.[2, 18] Under the microscope this line of junction is so thin that it renders the cementum almost non-existent; i.e., in a large number of normal teeth, the dentin already lacks an adequate cemental covering before being exposed by the detachment of the periodontal ligament.

Clinically, cervical hypersensitiveness indicates that the dentin is exposed, for cementum is devoid of any means for the transmission of pain,[12] and the periodontist does not need to be told the frequency of pain at the first stroke of the curetting instrument, even though there seems to be no break in the continuity of the tooth surface. On many others a definite break in the smooth continuity of the surface is detectable to the first passage of an exploring instrument under the gingivae, or visible as cervical decay or a wedge-shaped abrasion. On these, the cementum is already lost.

In the literature, photomicrographs of

complete loss of the cementum at the cemento-enamel junction are legion.[6, 13, 14] Examination of a large number of slides with Dr. Harold Keith Box[15] revealed an unexpectedly large number with either complete loss of or badly deteriorated cementum at the cemento-enamel junction, and farther toward the apex as well.

In other words, the dentin is already exposed in large numbers of teeth before the cemento-enamel junction is curetted and planed.

The second common teaching presupposes that all but the superficial outer layer of cementum is sufficiently hard and resistant that it will remain a permanent, or even a reasonably long-lived protective covering for the root surface, following careful surface curettage.[8] In our experience, this is not true. Cementum, devoid of epithelial and periodontal attachment, is a nonviable tissue and deteriorates. If reasonably healthy soft tissues continue to cover it, the deterioration is slow; in the presence of active inflammation, relatively rapid. If the soft tissues recede, exposing the cementum to the hazards of the mouth, deterioration is usually more rapid, even to extensive cervical caries,[16] though again, in a healthy mouth, the deterioration may be relatively slow. Exposed cemental surfaces are easy prey to abrasions such as toothbrushing. Denuded cemental surfaces are not sufficiently hard and resistant for a durable protective covering of an exposed root surface.[11, 16, 17, 18, 19, 20]

The third teaching questioned is that it is possible to curet even a normal, recently exposed cemento-enamel junction with a *sharp* instrument to a hard, smooth surface without removing the cementum in its entirety. To prove our contention, the following is a description of the findings in our studies on extracted teeth.

THE STUDY

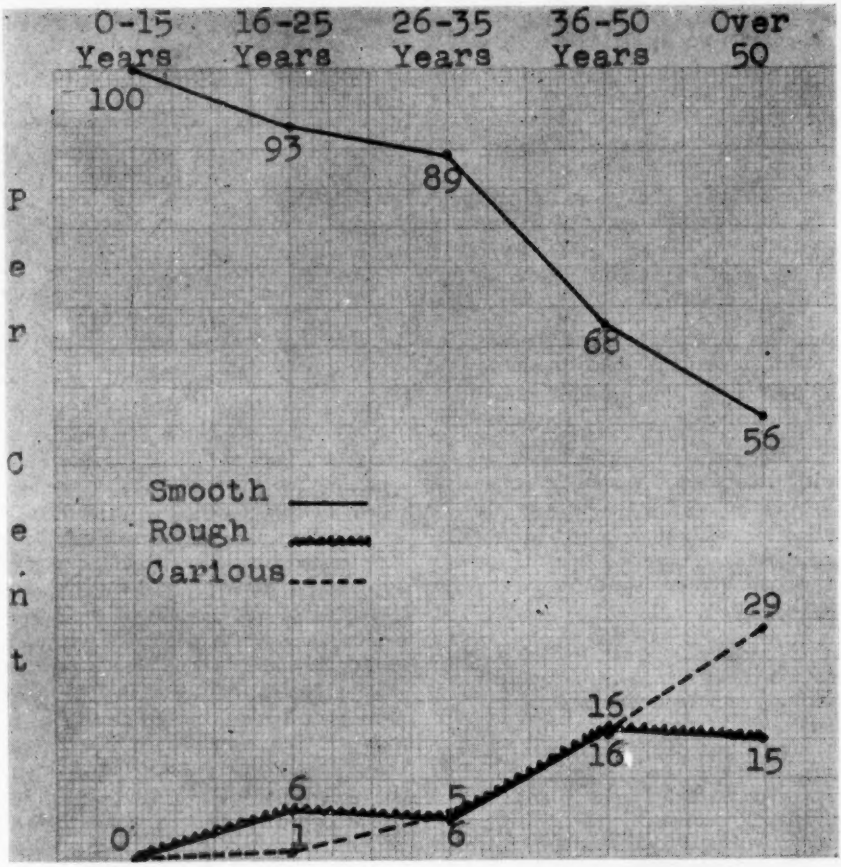
Five hundred extracted teeth, from the mouths of 312 patients were selected from the offices of 3 oral surgeons. All teeth were accepted as they appeared in routine sequence, except those where caries, restorations, or mechanical injury rendered them worthless for our purposes. All teeth from each patient were placed in a separate bottle of zephiran chloride solution, having previously ascertained that months in this solution seems to have no effect on the physical characteristics to be studied. It is more satisfactory to work on a wet tooth, for even a short drying changes the characteristic feel. On each bottle the surgeon noted the age, the sex, and the reason for extraction. The statistical subdivisions have been previously published.[21] Sufficient for this article is to state that each group and sex was represented in sufficient numbers to be significant.

Each tooth was photographed on each of its four surfaces, careful observations of the gross appearance of the cemento-enamel junction recorded, and measurements to 0.5 mm accuracy made of the greatest diameter at the cemento-enamel junction, both bucco-lingually and mesio-distally, for comparison with similar measurements after curettage.

A *sharp* instrument was then passed repeatedly over the cemento-enamel junction on every surface of every tooth, attempting to simulate the recommended procedures in subgingival curettage. At first we employed the more popular curets and planes, as Gracey's, Towner's, and McCall's, but soon found this too arduous, and being satisfied that the end results are the same, completed the series with a spoon-shaped Kingsley vulcanite scraper.

Recordings were made during the planing process as to the character of the surface: whether soft or hard; whether the scrapings were in the form of shavings, flakes, or powdery; and whether difficult to remove or comparatively easy.

Measurements through the greatest diameter at the cemento-enamel junction, both bucco-lingually and mesio-distally were then repeated and compared with those previously recorded. Photographs were



Graph 1. The visible characteristics of the cemento-enamel junction before curettage, classified in age groupings. (See text)

retaken in the identical position as before curettage, and the general character of the resulting pattern recorded.

THE CEMENTO-ENAMEL JUNCTION BEFORE CURETTAGE

Graph 1 depicts the findings from gross examination of the cemento-enamel junction, after calculus has been removed, if present, but before any curettage of the surface. Note that all teeth are macroscopically smooth and normal before 16, and that by 35 only 11% show roughening or caries. After 35, however, the incidence of visible deteriorations increases rapidly, until in teeth after 50, 44% are visibly roughened or carious.

When compared with previously published studies of the cervical enamel[21] one notes the general pattern the same, but about 20 years later in life, i.e., in the enamel studies, there was a 20% increase in the incidence of visible roughness and caries in the period 16-25 years; here, a corresponding increase during the period from 36-50 years. It would seem that there

is some deteriorating influence at or near the gingival margin, or within the pathologic crevice itself, active on the enamel in early life and on the cemento-enamel junction at a later period, as the gingival tissues become progressively detached from the root surface. In studies of the depth of the pathologic crevice,[1] a greatly increased rapidity of pocket formation was demonstrated during this same period. Surely this simultaneous increase in roughness and caries is not pure chance.

Studies of cementum and dentin to be published later will show a similar progressiveness of this unknown deteriorating influence.

Visible caries and roughnesses appear with about equal frequency before 50 years of age (Graph 1). After 50, there is a large increase in the frequency with which caries of the cemento-enamel junction is found, an observation which clinical practice readily verifies.

These observations seem to be strong corroborative evidence to the statement above, that denuded cemental surfaces are not sufficiently hard and resistant for a durable protective covering of an exposed root surface.

OBSERVATIONS DURING CURETTAGE

No teeth were found on which the repeated passage of a sharp instrument over the cemento-enamel junction failed to remove some surface. Only 4% of these cemento-enamel junction surfaces before the age of 25 showed a surface loss smaller than 0.5 mm. All others had an overall loss of diameter, both bucco-lingually and mesiodistally of 0.5 mm or over. After 26, less than a fraction of 1% of surfaces were found under the 0.5 mm minimum.

All of the teeth before 16 still had the periodontal ligament attached at the cemento-enamel junction and must be considered the normal at the present time.[1] Still we have 96% of these surfaces with a loss in diameter of 0.5 mm or over. As this is the total of two opposite surfaces, and observation during curettage seemed to justify the assumption that the early loss in these areas was approximately equal, we have a loss of at least 0.25 mm on each surface. This is 5 times the greatest estimated thickness of the cementum at the cemento-enamel junction.[22] Therefore, the cementum was removed in its entirety during the repeated passage of the curetting instrument.

Actually, the loss is much greater on most teeth than described above, and will be discussed in more detail in an article on the dentin during curettage.

On some teeth, particularly in the young, the feel to the curetting instrument is quite hard, and the material removed is quite fine and powdery; on most, the first stroke of the instrument encounters a softer surface, and the material removed is in the form of definite shavings (Figure 1), which softness gives way to an ever increasing hardness as curettage progresses; on many, the surface is definitely soft, even though no visible deterioration is present; when visible caries is present, continued planing carries us through the same sequence of very soft, to soft, to not quite hard, to hard, to the smooth hard feel to a sharp instrument that one finds in healthy enamel and dentin.

As in previously reported studies of the depth of the pathologic crevice,[1] and of the cervical enamel,[21] the labial/buccal surfaces presented the most frequent and the greatest degree of softening. The lingual was next in order, the mesial next, and the distal the least. For example, before 16 there were no teeth without visible and measurable loss on the buccal and on the lingual, whereas 25% of these same teeth presented a less than 0.5 mm loss on the mesial-distal diameter.

DISCUSSION

In studies of the cervical enamel,[21] we demonstrated that planing the surface with a sharp instrument resulted in a definite loss of enamel at the cemento-enamel junc-

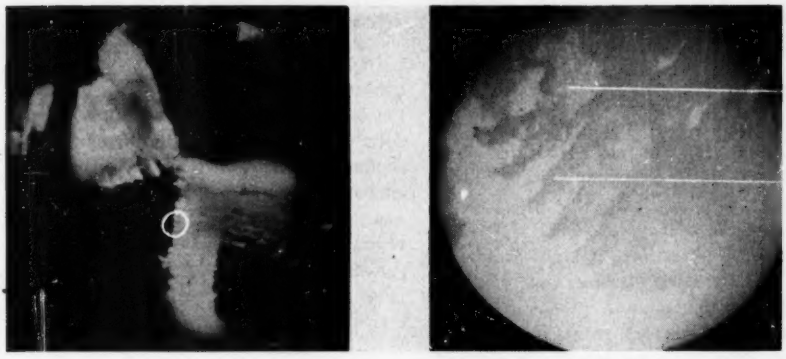


Fig. 1. A—Photomicrograph of shaving from cementum. B—Higher magnification of area circled in A. Note the worn-eaten deteriorated area at d, and the jagged, saw-tooth effect as s.

tion on most teeth. Here, the planing results in the loss of the cementum at the cemento-enamel junction in all teeth. Thus, if we follow most teachings and curet the surface of the root to a hard, smooth, "velvety" surface,[23] like "polished ivory,"[24] we have removed part of the enamel, and the cementum in its entirety. This violates a sacred dictum—that we must remove only the outer soft layer of cementum, containing the putrescent fiber ends;[8] that if we curet through the cementum, the healing of the pocket may not take place, and the denuded area will be permanently sensitive.[25, 26]

The teachings of G. V. Black[12] seem to have been forgotten: "In many cases the cementum of a pocket is considerably softened by the growth of acid forming microorganisms within the pocket. The acid dissolves the calcium salts from the cementum, softening it in the same manner in which dentin is softened by caries. Cementum in this condition is very easily removed with a sharp instrument."

H. K. Box[18] believes Hartzell's opinion that curettement through the second layer of cementum would expose the cement corpuscles of the third layer is erroneous, and that fortunately the fears of cemental infection through over-curettement are ungrounded. Also, that "exposure of lacunae and canaliculi with their contaminated cells and processes to long-standing infecfective inflammation in the adjacent soft tissues would certainly be a menace to the life of these minute particles of protoplasm. Box also believes that "it is beyond the human sense of touch to determine in curettement when the dentinal-cemental junction is reached, and because of its thinness, and other peculiarities, the cementum must of necessity be frequently removed in its entirety." He also considers the outer layer of dentin a compatible foundation from the biological standpoint.

Surely a tooth is not periodontally hopeless because of cervical hypersensitivity, cervical caries, and wedge-shaped abrasions, yet the dentin is exposed in all of these. Nor are the 10% of teeth lacking a cemental covering at the cemento-enamel junction doomed to hopeless periodontal disturbances when the soft tissue recession leaves this area unprotected. Dentin is perfectly capable of being exposed for years without visible change, except that in many it seems to become harder and shinier. Every dentist has seen innumerable cases in which the cervical enamel has been lost, which have remained unchanged for years;

many cases of teeth from which gold crowns have been removed and not replaced; many cases of exposed roots from which the gingivae have receded, with sufficient concavity of the root to leave no doubt but that the cementum has been lost. Even if the cementum were not removed during the first attempt at subgingival curettage, repeated planings would certainly remove it, and we are all aware that repeated planings and "removal of calculus" is common periodontal procedure. We have seen many of these on patients who have been under treatment for years by older practitioners, and interestingly enough, these are the teeth on which the gingival tissue is usually the most healthy.

It has been our experience that removal of the cementum at the cemento-enamel junction during curettage is not only inevitable, but also desirable. Failure to remove it and the deteriorated cervical enamel[21] results in failure to obtain the desired restoration of periodontal health. Ten years of making this a definite objective during curettage have convinced us of the advantages of this procedure. Sensitivity is more severe and more prolonged following partial removal of these soft surfaces than following complete removal; periodontal health is either not attained at all or is of very short duration following partial removal—quite certain and of long duration when entirely removed; cases which have resisted more conservative treatment, usually respond readily to this more radical approach. Even on an individual tooth, areas in which one has successfully removed all the softened structures, gradually return to a high degree of normalcy, while an area, be it ever so small, in which this has not been done, will remain chronically unhealthy—unhealthy enough that the patient will call the operator's attention to it as an uncomfortable area during mastication and toothbrushing; an area which bleeds easily; an area in which they repeatedly feel the need for floss or toothpick; and an area in which thorough curettage actually "feels good while it hurts."

In this article the discussion has been confined to the cemento-enamel junction; the cementum will be discussed at a later date. Complete removal of the deteriorated enamel and the cementum at the cemento-enamel junction is not all there is to treatment of pockets in which the bottom is beyond the immediate cemento-enamel junction area, nor is the cementum the only tissue involved. In very early periodontitis, however, in which the cemento-enamel junction has been recently exposed, removal of these superficial softened surfaces is frequently adequate treatment.

CONCLUSIONS

1. The dentin is already exposed in large numbers of teeth before the cemento-enamel junction is curetted and planed.

2. Denuded cemental surfaces are not sufficiently hard and resistant for a durable protective covering of an exposed root surface. Studies on extracted teeth, demonstrating increasing roughness and caries with increasing age provide some confirmation.

3. It is not possible to plane the cemento-enamel junction to a smooth, hard surface without removing the cementum in its entirety, for repeated passage of a sharp curetting instrument on 500 extracted teeth demonstrated a measurable amount of loss of surface much greater than the thickness of the cementum in this area.

4. The teaching that one must remove only the soft outer layer of cementum during curettage is discussed, with the conclusion that the removal of the cementum at the cemento-enamel junction is not only inevitable, but also desirable. Ten years' clinical observation is offered as supportive evidence.

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CALIFORNIA ACADEMY OF PERIODONTOLOGY

The new officers of the California Academy of Periodontology are: Dr. John H. Bajuk, President; Dr. Herbert E. Ward, Vice-President; Dr. Francis J. Fraher, Recording Secretary; Dr. Elmer G. Smith, Financial Secretary; and Dr. John Conroy, Treasurer. The Chairman of the Membership Committee is Dr. Robert Newton, and the Chairman of the Program Committee is Dr. Herbert E. Ward. Details regarding this organization may be obtained from Secretary Fraher.

Case Reports

Recurrent Herpetic Stomatitis Treated by Intradermal Injections of Influenza A & B Virus Vaccine

by C. KENNETH COLLINGS, B.S., M.A., D.D.S.,* *and*
CHARLES D. DUKES, B.S., M.A., PH.D.,** *Dallas, Texas*

MRS. E. B., 28 years old, came to the Baylor College of Dentistry clinic on January 31, 1951, complaining of large, extremely painful ulcers on the inside of her lips and cheeks. Examination of the oral mucosa revealed nine ulcers ranging in size from 6 to 15 millimeters in diameter. The ulcers were flat, faintly yellow on the surface, eroded into the tissue and surrounded by a narrow band of red, inflamed tissue. None of the ulcers had a true membranous covering. See Figs. 1 and 2.

A biopsy of one of the lesions was taken immediately and the laboratory reported the presence of intranuclear inclusion bodies, characteristic of Herpes simplex virus, in cells from the base of the ulcer. This finding confirmed the tentative diagnosis of Herpetic stomatitis based on the clinical examination and the history of the patient.

Mrs. B. stated that she had suffered from recurrent attacks of oral ulcers for about 15 years, which started about one year after the onset of menstruation at the age of 12. She could not recall that the occurrence of the ulcers coincided with her menstrual periods during her youth. The first attacks were mild and the ulcers were small but painful. As she passed through her teen-age years, the ulcers became progressively more severe, with an average duration of two weeks, but did not occur in any regular cycle.

At the age of 20 the patient married and shortly thereafter a very definite rhythm appeared in the recrudescences of the ulcers. Approximately one week prior to menstruation three or four small ulcers would appear and become progressively worse until menstruation started, at which time she would experience immediate relief. Normally the ulcers would disappear by the end of her menstrual period but would reappear invariably about one week before her next period. The only wide variation from this cyclic pattern reported by the patient was one severe episode previous to the present extending over a period of two or three months.

The only extensive periods when the patient had been completely free from Herpetic lesions was during her pregnancies. She has four living children, ages 7, 6, 4, and 2. She was pregnant for the fifth time last summer but had a miscarriage after three months, probably as a result of an illness. At the onset of each pregnancy, the patient experienced a "terrific flare-up" of ulcers, followed by a complete disappearance for the duration of the pregnancy. The patient stated that "once my mind was at ease that I was really pregnant, I would stop worrying about it and relax and they would stop." She also expressed her feeling that pregnancy was a welcome condition because it gave her relief from her ulcers, but for economic reasons she and her husband thought four children were enough. Following delivery, the ulcers recurred with the same cyclic pattern.

The parents of Mrs. B. are both native born and living. Her father, age 52, has been edentulous for about six years. He gives no history of Herpetic infection and his health is good. Her mother, age 50,

*Baylor University College of Dentistry.

**At present, Research Associate, Dept. of Plastic and Maxillofacial Surgery University of Texas, Galveston, Texas.

Fig. 1

Fig. 2

has some of her teeth and wears partial dentures. Her health is only fair, but the difficulty appears to be of nervous origin. She started having oral ulcers during her youth and suffered recurrent attacks similar to those described by her daughter. She does not remember that she had the same rhythmical exacerbations and remissions, but she does remember that with the onset of menopause at the age of 39, the condition gradually disappeared.

The patient has one sister, age 24, who is in good health and who has no history of oral ulcers.

Mrs. B. has had a rather characteristic medical history. At the age of 16, she consulted a physician who diagnosed her condition as an "acid stomach," and a dermatologist who treated the ulcers topically without effect. In 1939, a young physician became interested in her case and worked with her for about a year, approaching the problem from an endocrine standpoint. He prescribed a series of injections, and oral medicaments but no topical treatments. After one year of this therapy, he told her frankly that he could not help her.

For the next ten years, the patient did not seek medical assistance for the ulcers. Then, in 1950, she consulted a diagnostician, who opined that the ulcers were probably of endocrine origin. He gave her injections of progesterone until her fifth pregnancy occurred in July 1950, at which time they were discontinued. Following her miscarriage, in September, the ulcers returned and the patient consulted the diagnostician again, who resumed the progesterone therapy and referred her to Dr. S. R. Parks for periodontal consultation. Dr. Parks brought her to the Baylor Clinic because of the teaching possibilities of her case.

ORAL EXAMINATION

The occlusion is about average except for considerable vertical over-bite. The upper centrals are retruded and the upper laterals are crowded and extruded. The lower right cuspid and lower left lateral are also crowded and extruded. The lower right first molar is missing and has been replaced by a fixed bridge. The lower left second molar, lower right third molar and both upper third molars are missing. There are 15 amalgam and silicate restorations in her mouth.

The gingiva appeared healthy in the lower arch but, in the upper arch, there was a slight marginal gingivitis from the cuspid to the second molar region on each side. The tongue was smooth and of normal

size and color. There were shallow interproximal pockets of about 3 millimeters depth in the upper right and left and lower left quadrants.

Radiographic examination reveals three impacted third molars. No immediate operative dentistry is indicated. The bone appears moderately dense with about 3 or 4 millimeters of bone loss in upper right and left and lower left quadrants. The periodontal space is normal, with a clear lamina dura and cortical plate visible.

TREATMENT

When the patient was presented for treatment, she had been having a vicious attack of ulcers for over two weeks with only a slight remission when menstruation occurred and she was almost frantic with pain. Treatments were started immediately using smallpox vaccine.

Feb. 1, 1951—Smallpox vaccination.

Feb. 7

Feb. 14 At each of these dates there was a slight
Feb. 21 vaccinoid reaction present from the previous vaccination.

Feb. 28 The ulcers were not improved and the pa-
Mar. 7 tient was revaccinated.

Mar. 14 Slight vaccinoid reaction present. Ulcers unchanged. At this time it was decided that the smallpox vaccine was not producing the desired result and that influenza A & B virus vaccine should be substituted.

Mar. 14 An intradermal injection of 0.1 cc of influenza A & B vaccine, diluted 1:10 in sterile physiological saline was administered.

Mar. 21 Distinct, slightly indurated, hyperemic area was visible at the site of injection. Patient reported that about 72 hours after this treatment there was a distinct feeling of improvement in her mouth.
Ulcers much improved at this time. Intradermal injection of 0.1 cc of the vaccine diluted 1:5 administered.

Mar. 24 Hyperemic, indurated reaction was apparent at the site of injection. Ulcers were completely healed. Intradermal injection of 0.1 cc of vaccine diluted 1:2 given.

Mar. 27 Continued reaction was visible at site of injection. One painless ulcer was present, about 2 mm in diameter. Patient reported mild influenza symptoms. Intradermal injection of 0.05 cc of full-strength virus vaccine administered.

Mar. 31 Hyperemic reaction visible at point of previous injection. One small painless ulcer was present. Mild influenza symptoms were evident. Injection of 0.05 cc full strength vaccine given.

Apr. 4 Hyperemic reaction present. One very small painless ulcer observed. Patient, if in usual cycle, should have had several large ulcers at this time. Influenza symptoms were more distinct. Injection of 0.05 cc full strength vaccine given.

Apr. 7 Hyperemic reaction present. No ulcers. Influenza symptoms were present but mild. Injection of 0.05 cc full strength vaccine given.

Apr. 11 Hyperemic reaction present. No ulcers. Influenza symptoms present in mild form. Injection of 0.05 cc full strength vaccine given.

Apr. 18 Hyperemic reaction present. No ulcers observed. Influenza symptoms were clearly manifested. Patient stated that for past week she had experienced aching joints, low-grade headaches and some nasal congestion. She felt she was experiencing a light case of influenza. The patient apparently suffered a light systemic reaction following the last injection.

It was decided that the patient had become sensitized to the influenza virus and that it would be dangerous to maintain the injections. Treatments with the influenzo virus were thus discontinued after eight injections over a period of 28 days. Patient was asked to return at intervals for further observations.

Apr. 25 One small painless ulcer was found. She ordinarily would have had several at this time.

May 8 One small ulcer, painless, was present.

May 16 Two small ulcers, somewhat painful, were observed.

July 11 The patient reported that she had had only one or two small slightly painful ulcers, during the period between May 16 and June 27. At that date she had two very small ulcers which were only slightly painful. She was due to start her menstruation on July 2, but there was a delay of five days during which time she became extremely nervous and concerned. During this five day interval, the two ulcers became more severe and grew to about 8 mm in diameter. On July 7, the menstrual flow started and the ulcers immediately regressed and were much improved at the time of this examination.

July 17 Three small, painless ulcers were present. At this time, according to her earlier cyclic pattern, the patient should have been asymptomatic.

July 20 Patient had ten ulcers. Eight of them were only 2 or 3 mm across and were not painful. One occurring on the lingual frenulum was about 5 mm in diameter and was exceedingly painful, probably because of frequent motion during conversation. The other large one was on the inside of the lower lip and was about 12 mm in diameter and painful. The patient was under treatment by a physician at this time for a fungus infection occurring on her hands and on the vermilion line of her lower lip and for labyrinthitis. It seems reasonable to assume that her dermatophytic infection resulting in a lowered resistance may have been a strong factor in the appearance of the ulcers in her mouth. She was in the stage of her menstrual cycle where she ordinarily would have been free of ulcers.

July 27 At this time patient was away from Dallas and was under the care of a physician until September 1. Physician prescribed 250 mg. capsules of Chloromycetin every three hours for 36 hours. No improvement occurred.

July 30 Patient received Proteolac (Searle and Co.) I. M.

July 31 Intra-muscular Proteolac and intravenous Neo-Calgluconate (Sandoz).

Aug. 1 Proteolac alone.

Aug. 2 Proteolac and Neo-Calgluconate.

Aug. 3 Proteolac alone.

Aug. 4 Dermatophytic infection had cleared and patient had no oral ulcers.

Aug. 27 Two small dermatophytic lesions appeared on fingers. Proteolac and Neo-Calgluconate administered. No ulcers appeared at this time.

Oct. 10 Patient reported that her fungus infection had not reappeared and that she had been completely free of ulcers since Aug. 4.

DISCUSSION

Mrs. B. is one of the relatively small group of people who maintain a very delicate equilibrium with the virus of Herpes simplex. In her youth, previous to her marriage, this balance was stable with only irregular and infrequent attacks of ulcers. The endocrinal adjustments occurring at puberty probably initiated the appearance of the ulcers and, as the years passed, they slowly became progressively more severe and more frequent in their appearance.

Psychosomatic factors appear to influence the rhythmicity of occurrence. Mrs. B. is of a religious faith which does not condone the use of contraceptives. Following marriage, since the relative security of contraceptives was denied her, the fear of pregnancy developed prior to each menstruation provided sufficient mental agitation, with a consequent lowering of resistance, to precipitate the recrudescence of the ulcers. As soon as menstruation started, the worry was alleviated and the ulcers immediately began to heal. This may well account for the rhythm of their appearance and disappearance being established soon after her marriage.

During the time Mrs. B. has been under treatment at Baylor, there have been several other events which have produced a marked emotional effect upon the patient and a lowering of her resistance. At different times her small son was very seriously ill and had to undergo a splenectomy, her husband suffered a broken arm, and one of her other children was seriously ill for a time. With four children, the eldest of whom is only 7, there are undoubtedly many minor emotional crises each day, the total of which is sufficient to produce an almost constant state of nervous tension.

The treatments administered appear to have reduced greatly the number, size, and painfulness of the ulcers. The rhythm of their appearance appears to have been disrupted but this may not have been entirely the result of the treatments. In October, 1950, Mrs. B.'s husband had a vasotomy but the attending physician did not declare him to be entirely sterile until February, 1951. Her last and most vicious attack of ulcers started about January 15, 1951. Mrs. B. is now free of the worry of pregnancy and this factor is probably of great significance in the loss of regularity of ulceration. In July, when her menstrual period was five days late, she experienced the most severe exacerbation since treatment was initiated. The patient stated that she "didn't quite trust the operation and

so was not sure."

It cannot be said that Mrs. B. is "cured" of her recurrent Herpetic stomatitis although she has experienced several months of relief and attenuated outbreaks. It is quite likely that any illness or infection that she may contract will result in a full-scale recurrence of the ulcers by interfering with her resistance to the virus. She is walking the thin line between susceptibility and resistance. With good health and a minimum of emotional stress, she may stay relatively or entirely free of ulcers. However, any number of unfortunate events could easily return the patient to her original condition. It probably would be inadvisable for this patient to receive another series of influenza A & B virus vaccine injections due to the definite possibility of a systemic allergic reaction. If she can learn to lead a calm and equitable life and to minimize her daily worries, she may possibly be free or almost free of ulcers indefinitely. The future progress of the case will be watched with interest.

Acute Fulminating Suppurative Periodontitis

by WILLA YERETSKY, *Grand Rapids, Mich.*

MRS. A. is a white married woman aged 36. We first treated her fifteen years previously for an acute ulcero-membranous gingivitis. This responded promptly to treatment, leaving no permanent sequellae. The gingivae regained their normal level of attachment and there was no pocket formation. Following this episode the patient reported for prophylactic treatment at regular intervals during which there was no remarkable condition of any kind.

A year or two ago the patient moved to another city and placed herself in the care of a periodontist locally. Therefore, we did not see her for a year or more, at which time she requested attention urgently because of severe pain.

Examination revealed an acute peridental abscess of the lower left second molar, a recent extraction of the lower left first molar, an acute peridental abscess of the lower left second bicuspid, the lower right second bicuspid and the upper right first bicuspid. The gingival tissue around these areas was the typical swollen inflamed and painful type of an acute lateral infection and localized to these areas. Green, fluid pus was copiously expressed. Her temperature was 101° F. Laboratory findings showed an increase in her WBC to 10,000, and, in her sedimentation rate. Upon questioning, she stated that about two weeks previously she had had a severe pain which she felt was in the left first molar region. She consulted her local periodontist who X-rayed the region but saw nothing of any significance in the ray. He was unable to make a diagnosis and was unable to relieve her. The pain continued and became so intense that she no longer had any sense of localization. She then sought the help of an oral surgeon. He prescribed aureomycin. This gave no relief. Four days later, presumably on the basis that the pain had begun with the lower left first molar, the surgeon removed that tooth. Aside from the aureomycin four days previously and which was not continued up to the time of extraction, the patient was given no therapeutic dosage of any anti-biotic, nor was she given any immediately following the extraction. After this, she felt no surcease of pain whatever and felt increasingly ill. She attributed the continuing pain to the healing of the socket, but when two weeks had elapsed and she felt no better, she came into our office.

This patient was one whose oral hygiene was always excellent. She had no atrophic nor degenerative lesions. The problem was entirely that of a high grade, very acute and penetrating infection. Whether or not, one agrees with the Rosenow theory of the elective localization of bacteria, it was certain that in this patient's mouth, something had happened which made the peridental membrane, a previously strong and healthy tissue, susceptible to acute infective organisms. The organisms attacked the peridental membrane laterally, each abscess being obviously produced similarly and nearly simultaneously. There was a circumscribed area of deep red, extremely painful swelling about each involved tooth. These areas were confined to the buccal surfaces, the lingual being free.

Since the inflammatory process was caused by bacterial invasion and not foreign deposits such as calculus, nothing would be gained by instrumentation of any kind. Moreover, undue manipulation could be responsible for a generalized increased bacteremia with results that could be most unpleasant.

It was therefore decided to use the Vastine method of rubber cup suction by hand. A rubber cup was filled with tincture of metaphen and gentle suction applied over each infected area. The entire oral cavity was also irrigated with tincture of metaphen. The patient was dismissed with a prescription for a 1:1000 potassium permanganate solution to use as a mouthwash every three hours at home.

While her physical depression could certainly be accounted for by her multiple abscesses, it was nevertheless very important that we be sure there were no other systemic complications. So from our office, the patient was at once referred to a physician. At that examination he found nothing significant except the oral symptoms.

The following day, the oral lesions showed a very marked improvement but the patient felt no better. She was, therefore, hospitalized. An X-Ray of her chest showed an area of pneumonia. Her recovery, under the usual anti-biotics, was complete and uneventful. The oral lesions subsided completely within three days from our initial treatment. There are no persistent pockets. Attachment is normal and complete with complete regeneration of the alveolar process and membrane.

In the last three years, we have had four similar cases. All responded to the Vastine method of rubber cup suction with tincture of metaphen. None had any persistent pockets following recovery and none has had a recurrence.

Mrs. A. is the only patient who developed any localized systemic symptoms apart from those in the mouth and it is therefore presumed that a sufficient bacteremia was set up, following the extraction in an acutely infected area, to cause a pneumonia.

We are inclined to agree with the hypothesis that the anti-biotics so generally in use, have caused a reduction in the cocci produced infections and are allowing virus strains of organisms to become pathogenic. Whether or not, the chief offenders in the case described were of the virus type, we are not prepared to say—No cultures were studied.

In conclusion, may we emphasize that it is most important first of all to make a very careful differential diagnosis of any localized abscess or abscesses. Secondly, every effort must be made in treatment, to avoid trauma of the infected areas. Even a slight trauma may produce a bacteremia which may be sufficient to produce a septicemia.

Editorial

Book Reviews

It is customary for many journals to publish reviews of new books, particularly those which should interest its particular group of readers. We have received three such books:

The Temporomandibular Joint. *Edited by* Bernard G. Sarnat, Professor and Head of the Department of Oral and Maxillofacial Surgery, College of Dentistry, University of Illinois, with Allan G. Brodie, Harry Sicher, John R. Thompson, Joseph P. Weinmann, and Arnold Zimmerman contributing authors. These men have reviewed and analyzed the literature on the temporomandibular joint from the point of view of both the basic and clinical sciences. The book is beautifully published by The Charles C Thomas Publishers of Springfield, Illinois, (1951) and includes over 50 illustrations and 148 pages. Everyone interested in the temporomandibular joint will want to study this book.

Dental Chronology. *By* Hermann Prinz, Emeritus Professor of Pharmacology and Lecturer, The Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania. This interesting volume of 188 pages is filled with an interesting summary of important historical events or landmarks, from the most ancient information up to 1944. Also included is an appendix containing much valuable historical information. Dentists interested in history will find this book, published by Lea and Febiger (1945), most helpful.

Pharmacology and Pharmaco-Therapeutics for Dentists. *By* W. H. O. McGahee and M. W. Green. This is the fourth edition of this highly respected textbook, expanded and modernized to make it a valuable reference book for dentists as well as a text for students. The discussions of the fluorides, sulfonamides, arsenicals, dentifrices, and antibiotics present the latest viewpoints on these important materials. The book is published by Blakiston Company.

Now the question this Editor wants to have answered is this: Do the readers of the Journal want to have critical book *reviews* included, or are book *notices* such as these above more desirable? In other words, do readers want to be just informed of the existence of books of interest to the periodontist, or do they prefer a critical analysis of those books? Since this is *your* Journal, we await instructions.

President's Address

By HUNTER S. ALLEN, *Birmingham, Alabama, President 1950-51*

A SIGNAL compliment was paid me and a great honor bestowed when I was designated President of this Academy. In addition, you presented a challenge of service to our profession through this specialized group, whose contributions are the best in dentistry. Even though poorly equipped, the duties and responsibilities of the office have been pleasantly and easily discharged. This is due to the splendid encouragement and assistance of everyone in position to share the load.

I would like to thank publicly the many who have supported me so generously and efficiently the past year. A heart-felt gratitude goes to those participating in the activities of the Academy, particularly: members of various committees and fellow officers. They have fostered the pervading spirit of fellowship, conserved our resources, and promoted educational advantages for which we have been acclaimed. It is you who make my presidency such an appreciated distinction.

We do not represent a new specialty in medicine or even a recent phase of dental practice. History records ancient study and treatment of periodontal lesions. Then, when medical knowledge was hopelessly inadequate, such diseases were referred to as "loose teeth" and "bleeding gums." Today our nomenclature, though not adequate, is more expressive of various types and progress of these morbidities. Since the dental attention given Rameses II, the recognition and treatment of periodontal disorders by Hippocrates, and years later, Pliny, we have advanced in ability to identify them and institute proper therapy; even that of a misplaced confidence in drugs, antibiotics, vitamins and more palatable mouth washes than animal excretions. Many highly endorsed healing agents of today will become the folly of tomorrow. This is progress!

Not too long ago periodontal diseases had but one classification, "Scurvy of the Gums," and those so afflicted were without hope of cure. It was not until 1867 that a responsible authority announced successful treatment. In the summer of that year Dr. John M. Riggs conducted a clinic before the Connecticut Valley Dental Association, demonstrating instruments of personal design, for removal of deposits from necks and roots of teeth. These, though crude compared with present patterns, and painful in application, produced remarkable results. Thus began the serious use of instruments and their gradual refinement. Dr. William J. Younger was the first to develop instruments so delicate they could be introduced into pockets for removal of deposits without injury to gingival tissue. Dr. T. Sidney Smith, a devout exponent of conservative subgingival curretage, used these with eminent success, to the point of possible vital reattachment. Not until 1896, however, did Dr. Eugene S. Talbot publish what is regarded as the first histopathologic study of inflammatory processes of the gingivae. Dr. Gottlieb began his studies in 1912 and since has made numerous contributions to our knowledge of periodontal tissues in health and disease.

The point I would make, in this recital of history, is that although much technical skill has been developed it was necessary for these histopathological findings to be presented before a comprehensive understanding of the disease and its etiology could be determined.

The normal development of the science of saving teeth was interrupted by Rosenow's theory of focal infection. According to his pronouncement teeth were responsible for so many systemic ailments that all edentulous people should be blessed with longevity. This was accepted by both medi-

*Read at the Thirty-fifth Annual Meeting of the American Academy of Periodontology, Oct. 11, 1951 at Washington, D.C.

cal and dental men as an easy explanation of their failures in diagnosis and treatment. The hypothesis sought a conclusion without consideration of natural processes, such as progressive deterioration and repair, wherein impairment is halted and recovery begins.

The situation was ripe for explosion when the Focal Infection idea was introduced. Black had already reported 13% of 600 patients between ages 20 to 24 and 88% of those over 50, afflicted with some form of periodontal disease. The two announcements served to emphasize the mental concept of the incurability of "pyorrhea," a name erroneously given all such morbidities. Under the circumstances it has been difficult to convince laymen, physicians and dentists that an afflicted tooth should not be removed to remedy a malady which escapes definite diagnosis. Fortunately the reaction to such an absurd fallacy is resounding in this Academy, which has as its prime consideration dental conservation.

In fact the American Academy of Periodontology owes its birth to a group of conservationists, who convened in Cleveland, Ohio, 1914, to organize the first Academy in dentistry. Their distinct purpose then, as now, was that of restoring periodontal tissues to health and maintaining them in such a state. No one expected of it a Phoenix of fable, fully grown. They were dedicated to a gradual and sure development without maturity. Today, we, who follow their precepts, know the tedium, toil and tragedy of growth and are proud to have a part in the evolution.

One of the earlier recognitions was the need of proper nomenclature to describe tissues involved and their reaction to pathologic changes. Since the term pyorrhea was being used all inclusively to indicate periodontal diseases, it became imperative to adopt a specific terminology. Hence the word periodontoclasia was introduced. This has been followed by more pronounced efforts and with better results until now when we have a fair foundation upon which to build a precise language. Our vocabulary continues to be too limited to serve as a comprehensive guide for those in this specialized study and practice. Since this is an indication of unfinished business I strongly urge the Committee on Nomenclature to work as a unit to hasten an acceptable report.

In periodontia there are today four distinct groups of conditions with which to deal, as the late Dr. John T. O'Rourke stated:

1. Those of purely local origin.

2. Those which are entirely oral manifestations of metabolic disorder, infectious disease, blood dyscrasias, nutritional deficiencies, and others.

3. Those which represent a fusion of both conditions of local and systemic origin.

4. Those which are systemic manifestations, as a whole or in part, of oral diseases, deficiency or disorder.

These different manifestations appear as the result of structural changes produced by one or several factors. It does not seem practical, then, to attempt the standardization of a treatment technique in periodontia.

The American Dental Association is beginning to recognize the importance of the etiology and treatment of periodontal injury. This is interesting but we should realize it is not the full embrace of the basic principles of periodontia. Education must precede understanding and accomplishment. Such education necessarily should start in schools devoted to dental instruction. We are amazed and discouraged by the failure of our dental schools to offer appropriate facilities for graduate and post-graduate study in periodontia. This present status is all the more regrettable because it prevents the wide dissemination of knowledge we now have, just when and where it would do the greatest good.

A survey of 41 schools, through questionnaires, by the Committee on Undergraduate and Post-graduate Study, of the

A. A. of P., reveals the fact that 15 have separate departments of periodontia; 12 include it with oral medicine or endodontia; 6 with operative dentistry; 4 with oral pathology.

The committe states: "We believe, however, that generally speaking periodontia can be better given as a separate course or more logically associated with oral medicine, oral pathology, oral surgery, or endodontia, than with operative dentistry."

Without proper training there can be no appreciation of advances in treatment and cure of baffling diseases. Dentists not qualified to contend with persistent periodontal lessions especially the pocket forming type, are those who pronounce it incurable.

We should more pronouncedly encourage our dental schools to institute adequate instruction in periodontia. Without such educational facilities there should be no A, or even B, classification. The institution is the best source of comprehensive instruction and emphasis. As Dean Maynard Hine has stated "One cannot debate the point that periodontia must receive more emphasis in dental education."

Concisely stated, the purpose of this Academy, as I see it, is: To help those seriously interested in periodontia grow in capability and capacity to render a real health service.

We are the American Academy of Periodontology. You and I are its adjuncts, or emissaries, who together make the composite. We are its existence and are responsible for its justification. In practice we are individuals and become personal representatives of this Academy in our contacts with the public. The organization will always be judged by personal and professional deportment of its members. Because of this, distinct care must be exercised in the selection of representatives. Membership should not be granted those who seek such recognition for ulterior motives. Proper qualifications must be the supreme consideration for admittance to our group. Sturdy growth depends upon those who bring to us a contribution of talent in exchange for advantages we offer. Even the hardy oak can be destroyed by the mistletoe which lives upon it. Stunted existence is too great a price to pay for decoration.

Our entrance door is now closed while we re-examine applications of those seeking admission and clarify the perplexing problem of qualifications. With a large and unscreened membership, as Dr. Dickson Bell observed: "This Academy would become just another dental society."

We bear the name Academy because we are an association of adepts and advanced students for the promotion of science and art in a chosen field. We must keep it that way.

During the year our Committee on Revision of Constitution and By-Laws has made important studies and prepared revisions to be presented for consideration in St. Louis. The present Constitution delays for two years final decision on important changes. We need a document which encourages, rather than hinders, progress. Some provisions are being deleted from the Constitution and recommended for placement in the By-Laws to facilitate speedy consideration and action,—even during an annual meeting. The purpose, of course, is to create a modern instrument of government.

The Academy has as its mission the conservation of surrounding and supporting tissues of teeth and the dissemination of knowledge incident to its importance and possibility. This is the spirit I have endeavored to perpetuate. For your generous support I express my great appreciation and heartfelt thanks.

Announcements

PERIODONTICS

DR. JACOBY T. ROTHNER AND STAFF

The objective of this course is to present *modern* concepts of Periodontics to the general practitioner of Dentistry. The course will include physiology and pathology of the tissues involved in the disease; the relationship of medicine, laboratory findings and bacteriology. The approach to etiology, diagnosis and treatment of the disease of the supporting tissues will be of a practical nature. There will be laboratory practice and clinical demonstration in occlusion, instrumentation and home care. Authorities in associated fields will take part in the instruction, and the whole picture will constantly be correlated.

Instruction will be by lecture, clinical demonstration, laboratory practice and open discussion.

Course starts Thursday, February 7, 1952 and
continues for seven successive Thursdays from

9:00 A. M. to 4:00 P. M.

Fee.....$175.00

This fee includes a $25.00 set of periodontal instruments.

Course limited to fifteen.

1951-52 STUDENT ESSAY CONTEST OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

To promote interest in periodontia among undergradute students, the American Academy of Periodontology is sponsoring an annual essay contest. This contest is in two general parts:

1. *Local Contest in each school.* Each year the American Academy of Periodontology wishes to award a year's subscription to the Journal of Periodontology to the undergraduate student who writes the best paper on periodontia in each college. Each faculty of each college can make its own rules and regulations and select the winning paper. The winner's name should be sent to the Editor of the Journal before May 15 of each year.

2. *National Contest.* Each year one paper from each school can be submitted to the Undergraduate and Postgraduate Education Committee of the American Academy of Periodontology on or before April 15. The three best papers will be awarded the following prizes:

First Prize—$50
Second Prize—$30
Third Prize—$20

These papers or abstracts of them may be published in the Journal of Periodontology as the best undergraduate papers submitted in the year.

PRIZE FOR RESEARCH OFFERED

The American Academy of Periodontology offers a prize of $100 for the outstanding report submitted on original research work in the field of periodontology. This paper can be written by an undergraduate student, graduate student, member of a dental school faculty, or practicing dentist.

Papers submitted should be sent to Dr. Robert G. Kesel, Chairman of the Committee on Scientific Investigation, 808 S. Wood St., Chicago by June 15, 1952.

COLORADO RESEARCH FOUNDATION ANNOUNCES TWO COURSES FOR MARCH, 1952

1. Theory and Practice of Periodontia—March 10-14
2. Histopathology of the Oral Cavity—March 16-20

The Theory and Practice course will consist of lectures and demonstrations as follows:

Lectures:	The epithelial attachment, Gingival sulcus and pocket
	The normal gingiva, Periodontal membrane
	Alveolar bone, Cementum
	Biology and Pathology of bone
	Systemic aspects of periodontal diseases
	Tissue changes due to trauma
	The problem of occlusion in periodontal practice
Demonstrations:	Prophylaxis, Home-care technique
	Gingivectomy, Post-operative treatment
	Registration of hinge axis, Mounting of models in articulator
	Occlusal adjustment

Class starts Monday, March 10th at 9:00 A. M. and ends Friday at 9:00 P. M.

Fee: $200.00

The Histopathology course will be conducted by J. P. Weinmann, Professor of Oral Pathology—University of Illinois Dental School—assisted by Dr. Orban.

Lectures & Microscopic Work	Pathologic conditions associated with developmental disturbances of the face: Clefts, cysts, hard structures of teeth
	Bone pathology, Pulp and periapical disturbances, tooth fractures
	Osteitis, Osteomyelitis
	Oral manifestations of systemic diseases: endocrine, nutritional
	Oral manifestations of dermatoses
	Tumors of the oral cavity: Benign, malignant, odontogenic
	Pathology of the temporo-mandibular joint

Class starts Sunday, March 16th at 9:00 A. M. and ends Thursday at 9:00 P. M. This unusual arrangement is made because of the meeting of the International Assoc. for Dental Research which opens in Colorado Springs on Friday, March 21st.

Fee $250.00

THE THIRD ANNUAL BERKSHIRE CONFERENCE IN PERIODONTOLOGY AND ORAL PATHOLOGY

The Division of Graduate and Post-Graduate Studies of Tufts College Dental School will conduct the Third Annual Berkshire Conference in Periodontology and Oral Pathology at Eastover, a resort hotel in Lenox, Massachusetts, June 15 to June 19, 1952. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities, with afternoons open for recreation. A special activities program is scheduled for the ladies. Tuition—$100.00. Room, board and incidentals—$70.00 per person. For futher information regarding this conference write: The Berkshire Conference, Tufts College Dental School, 136 Harrison Avenue, Boston 11, Massachusetts.

THE AMERICAN ACADEMY OF ORAL PATHOLOGY

will hold its Annual Meeting in Chicago,
at the Stevens Hotel, on February 3, 1952.

The following program will be presented:

9:00 A. M. — Histopathology of Various Gingival Enlargements,
Sigmund Ramfjord, University of Michigan,

10:00 A. M. — A Discussion of Dental Anomalies,
Myron Aisenberg, University of Baltimore,

11:00 A. M. — Dermatological Diseases Affecting the Oral Cavity,
Lester Cahn, Columbia University,

2:00 P. M. — Physiology of Bone,
Lent Johnson, Armed Forces Institute of Pathology,

3:00 P. M. — Bone Behavior in Periodontal Diseases,
Joseph P. Weinmann, University of Illinois,

4:00 P. M. — A Study of Oral Carcinoma,
Joseph L. Bernier, Armed Forces Institute of Pathology.

This is not a closed meeting and all interested dentists are invited to attend this program of the American Academy of Oral Pathology.

Dr. Joseph L. Bernier, Colonel, Dental Corps, United States Army, has been designated to supervise the activities of the American Academy of Periodontology at the meeting of the 11th International Dental Congress to be held in London, England, next July. Any members of the Academy who are attending the meeting and who wish to present table clinics are encouraged to do so. Anyone interested should contact Col. Bernier at the Armed Forces Institute of Pathology, Washington 25, D.C., at once.

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE
NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The periodontia Department of New York University College of Dentistry, announces its full time course in Periodontia and Oral Medicine for one academic year, September 22nd, 1952 to May 22nd, 1953, leading to a certificate. The course is limited to four. Half-time two year courses are also given as well as a three weeks refresher course beginning June 2nd 1952. All courses are under the direct supervision of Dr. Samuel Charles Miller.

For information concerning the above or courses in other phases of dentistry write to Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N.Y.

THE THIRD MID-ATLANTIC STATES SEMINAR IN ORAL MEDICINE

11 to 16 May 1952

The Postgraduate Courses of the School of Dentistry of the University of Pennsylvania is offering the Third Mid-Atlantic States Seminar in Oral Medicine at Skytop Club in Pennsylvania's famous Pocono Mountains, May 11-16, 1952. The general theme of the third seminar is concerned with the anatomy, function and efficiency of the masticatory apparatus and the application of this knowledge to clinical problems. An authoritative Faculty has been selected and the contribution of each participant has been integrated for the most effective presentation.

The clinicians include Dr. Oscar V. Batson on an anatomic review of the muscles of mastication and the temporomandibular joint; Dr. William Branstad on diagnosis and treatment of minor abnormalities of the temporomandibular joint; Dr. Henry M. Goldman on application of the knowledge of abnormalities of the chewing apparatus as a cause of periodontal disease; Dr. Ernest R. Granger on recording condylar path and centric relation; the significance of this information in restorative and prosthetic dentistry; Dr. Richard S. Manly on the efficiency of the chewing apparatus; and Dr. Robert E. Moyers on relation of abnormalities of the chewing apparatus to orthodontic problems.

Attendance for participation is limited to members in good standing of the A.D.A., A.M.A., or similar or comparable groups. The fee for this seminar, including registration, lodging and meals for five days is $250.00. Veterans who can furnish a satisfactory Certificate of Eligibility and Entitlement will receive a credit of $150.00 toward the total fee. Enrollment is limited. Applications will be accepted in the order they are received.

For further information write—Dr. Lester W. Burket, Director, Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia, 4, Pennsylvania.

PERIODONTIA COURSE AT BETH ISRAEL HOSPITAL

The Beth Israel Hospital of Boston, Massachusetts, announces a two-weeks course in Periodontia, under the direction of Dr. Henry M. Goldman, from March 3 to 14, 1952.

Instruction will be carried on by clinics, lectures, demonstrations and motion pictures. The fee is $300.

Anyone wishing to apply kindly write to Dr. Henry M. Goldman, Beth Israel Hospital, Boston, Mass.

PERIODONTIA IN GENERAL PRACTICE

On Thursday, February 7, 1952, immediately following the February Midwinter Meeting of the Chicago Dental Society, the University of Illinois College of Dentistry is once more offering a one day course in periodontia.

The subject this year is Periodontia in General Practice and will again be given in the dental college at 808 S. Wood Street. If enrollment is large enough, the course will be presented by means of television.

Several outstanding men on the faculty of this course will present a solution to the many problems with which general practitioners are faced in the treatment of periodontal disease.

Especially featured will be a new and unusual approach to the study of occlusion. This should be of considerable interest to the periodontist as well as the dentist in general practice.

Those interested, should write Saul Levy, D.D.S., Coordinator Postgraduate Studies, 808 S. Wood Street, Chicago 12, Illinois.

COUNCIL OF ACADEMY TO MEET

Dr. Clark E. Chamberlain, Secretary of the American Academy, announced that the Council of the A. A. P. will meet in Chicago, February 2, 1952.

Letter to the Editor

Importance of Nutrition Stressed

Dear Dr. Hine:

I have read with interest Dr. Paul Boyle's article on the experimental evidence of systemic influence on the development of periodontal disease. Dr. Boyle's article did not go far enough.

For many years the Extension Courses of the University of California in conjunction with the Southern California State Dental Association have given us post graduate courses in endocrinology, bone pathology, hematology, psychosomatic medicine, nutrition and X-ray interpretation and diagnosis. Dr. Boyle's article touched on the influence of Vitamin A, Vitamin D, Vitamin C and endocrine secretions in regard to oral health. As you know, nutrition is not taught in the medical and dental schools of this country. This fact has grave national significance. Since wheat germ has been removed from our flour we are faced with a national B Complex deficiency. It is well known that several factors in the B Complex are essential to the health of the oral mucosa. The Periodontist should be concerned with the health of all cells of the body as they are all reflected in the oral cavity. The health of the twenty-seven thousand billion cells that comprise the gastro intestinal tract is of interest to us because the mouth is one end of this tract. The taking of laxatives not only interferes with food absorption but may act as irritants and predisposed to gastro intestinal carcimona. Nutrition optimum in all respects will restore and maintain gastro intestinal health.

Many patients present themselves with periodontosis within the normal weight range but a Diet Analysis will disclose that some of these people are maintaining their weight on from nine hundred to twelve hundred calories. Most patients that I see in Southern California have a low metabolic rate. Optimum nutrition will build up endocrine function to a more normal rate in these patients. The taking of thyroid by a patient over a long period of time will atrophy the thyroid gland. It is needless to say that these low caloric diets are gravely deficient in nutrients necessary for oral health. The Blood Picture should be of interest to the Periodontist; the calcium and phosphorus and its ratio in the blood, the cholesterol, fasting sugar, the red cell count, hemoglobin and differential white cell count. I have a physician run a Blood Picture before I start treatment and do nutritional correction. A recheck is made later on items that are out of balance in the Blood Picture. The cholesterol, sugar, calcium and phosphorus levels in the blood are in no way related to the amount of these items in the dietary. Cholesterol can be reduced with a high cholesterol diet. Blood sugar can be raised or lowered with optimum nutrition. This goes back to the nourishment of the endocrine glands and the liver. Tension which is cumulative and subconscious tends to cause vasoconstriction and reduces the blood supply to the various cells. Emotional upsets which are reflected in our gastro intestinal tract interfere with food absorption and proper endocrine function; therefore, the patient should be instructed in the necessity of acquiring emotional stability and a positive outlook on life and its influence on the body's economy. A patient on optimum nutrition has a better chance to put these suggestions into operation than a poorly nourished one.

Dr. William Albrecht of the University of Missouri has pointed out many times the significance of soil depletion and its relation to our national malnutrition. Dr. Weston A. Price's classical contribution to mankind was the fact that he visited the primitive people of the world to find out why they were not subject to degenerative diseases and not why civilized people were. Diabetics are told what not to eat but are

rarely put on an optimum nutritional program. A woman during pregnancy should have very optimum nutrition. Nausea, constipation and miscarriages in the first three months can be controlled by optimum nutrition. Lactation can also be improved. This is very seldom looked into and there is no reason why pregnancy should damage oral health. Faulty reducing diets that women indulge in cause a great deal of oral damage. Reducing diets optimum in every respect to rebuild endocrine function, reduce weight and restore oral health should be part of the armamentarium of the Periodontist. Recent research has pointed out that during the male and female menopause, particularly with indoor workers, the excretion of calcium in many cases is greatly increased. The Periodontist should compensate for this in the daily nutrition of his patient.

The effort of the dental profession to use fluorine and other substances to protect the enamel of the teeth so that poor nutrition may be indulged in to build unhealthy bodies for our youth is certainly an approach from the wrong direction. The recent statement of our Draft figures that 60% of our youth were being turned down is alarming to say the least. The increase in consumption of refined carbohydrates by this group is reflected in the Draft figures. In 1947 I saw a colored motion picture of Deaf Smith County (the County without a toothache). At the end of his talk the dentist who brought the picture here stated in passing that they had had a 7% turndown in the Draft of their youth for World War II. I told him at that time that the lack of tooth decay was merely incidental; the fine top soil and the good products grown there produced healthy bodies. In this day of physical softness people do not consume much over two thousand calories daily in Southern California. With the increased depletion of our soils and refinement and packaging of our foods it is necessary to supplement the American dietary with natural vitamins and minerals to make it optimum to maintain cell integrity. Natural A and D, B Complex and kelp for its mineral content, particularly collodial iodine, are necessary. It is needless to point out that the daily dietary should include optimum amounts of animal protein, meat, fish, eggs, milk and cheese, so that as the cells die, the building blocks for new cells from the ten essential amino acids are always present in optimum amounts.

Several years ago through about four issues of our Journal a controversy raged as to whether or not to brush the gums. As our foods become softer it becomes more necessary that the gums are included in brushing and that inter-proximal gum stimulation be taught. The health of any cell, epithelial, connective, nerve or bone is dependent upon the blood stream for all the nutrients and endocrine secretions essential to its health. Good circulation is necessary for cell integrity. Irritation and stagnation should be removed. As nearly as possible each tooth root should be allowed to carry its share of oral stress.

Periodontia is truly the one specialty of medicine and dentistry that should be entirely preventive because we are interested in the patient retaining their teeth and all factors necessary to accomplish this end are the dentist's responsibility. The "Science" magazine, published weekly by the American Association for Advancement of Science, from time to time contains "Gems of Nutritional Research" which are important to the Periodontist.

I realize that many statements have been made in this paper seemingly without substantiation. I have studied and practiced clinical nutrition for eighteen years. All the teachers I have studied under have gotten their material from the Bio-chemists connected with the Research Departments of the Universities throughout this country. Clinically we have been able to substantiate their findings.—Alex J. Linck, D.D.S., 1052 W. 6th St., Los Angeles 14, Calif.